ANNALS
OF
OTOLOGY, RHINOLOGY
AND
LARYNGOLOGY

VOL. 59 SEPTEMBER, 1950 No. 3

LV

BASILAR SKULL FRACTURES

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AUGUSTA, MAINE

Fractures of the base of the skull are here discussed under three sections: Surgical anatomy, symptomatology and treatment.

SURGICAL ANATOMY

The skull is composed of three layers—an outer and an inner solid table with a layer of diploetic bone serving as a cushion between the two. The diploetic layer varies greatly in thickness, and disappears entirely in the squama of the temporal bone and frequently in the posterior fossa. A moderate blow frequently fractures only the inner table because the outer table is supported by the diploetic layer and holds more firmly; and also because the inner table is thinner and has a shorter radius of curvature. The external layer averages 1.5 mm, and the inner 0.5 mm in thickness.

The skull is subjected to many blows of varying intensity and yet is rarely fractured, due to a number of factors which influence the effects of trauma. Some of the anatomical features of the head which tend to minimize the effects of blows or other trauma may be listed briefly as follows: (Pancoast[1])

1. The thickness, density and mobility of the scalp, especially the outer three layers, which move as a unit upon the loose underlying subaponeurotic layer.

2. The dome-like contour of the superior region of the skull. The shape causes blows to glance off, and the arching creates strength.

3. The mobility of the head in its entirety upon the spinal column, in rotary, lateral and fore and aft movements.

4. The number of bones, which tends to break up the intensity of the blow.

5. The several sutures, which interrupt the transmission of the force.

6. The intersutural membranes, which tend to absorb some of the force.

7. The sutural overlapping of some bones, such as the parietal by the squamous portion of the temporal, and the alternate overlap between the parietals and frontal.

8. The presence of structural braces; (a) from the crista galli to the internal occipital protuberance, (b) the temporal lines or ridges from orbits to the mastoids, (c) bilaterally from the external occipital protuberance to the foramen magnum, (d) from mastoid, (e) from the nasion or root of the nose to the zygomatics, (f) a number of secondary braces on the inner aspect of the skull.

9. Bones arranged as buttresses, such as the processes of the zygomatic and temporal bones and the great wings of the sphenoids.

The anterior fossa shows in the midline a free, heavy, sagittal ridge to which is attached the falx cerebri. Occasionally, this ridge is quite pronounced and hence affords considerable support. Where it terminates in the region of the cribriform plate, it is again reinforced by the crista galli. Arching above on either side one finds the orbital portion of the frontal bone. As additional support the anterior fossa is strengthened by the strong lesser wings of the sphenoid which terminate medially in the anterior clinoids. Laterally, the support is gained by the angular process of the frontal bone.

The obviously weak area of the anterior fossa is the thin cribriform plate and it is this portion of the anterior fossa which is most frequently fractured.

The middle cranial fossa is strongly supported by the heavy petrous bone whose apex terminates in the posterior clinoid process. The great weakness of the middle fossa lies in the number of large foramina directly anterior to the petrous pyramid: namely, the foramina, spinosum, rotundum and ovale. Because of these foramina, a fracture usually occurs directly anterior to the petrous bone.

The posterior cranial fossa has two sturdy supports; the firm bony rim of the foramen magnum, and the occipital proturberance. The floor lying between these is the point of weakness and is the basal point of fracture.

The two factors which cause fractures of the base are in an external force applied directly, or transmission of the force to the base from a blow delivered elsewhere, such as the vault. The external force can be from a number of sources; such as a blow on the chin forcing the condyle of the mandible upward, or a fall on the feet or buttocks which jams the occipital condyles against the atlas, or the penetrating foreign body wound in the orbit or mouth.

The dissemination of the force has been the subject of several theories and much discussion. Rawlings[2] has taken issue with the Aran theory of radiation, i.e., that fractures of the base result as extension from fractures of the vault, with the fracture following the shortest anatomic route. Rawlings believes that 30% of the fractures of the base occur in this fashion, but that the weak areas, which we have discussed, are selected and the stronger area not affected except in the most severe trauma where the fracture extends to the base irrespective of the varying elements of strength.

Almost any standard textbook propounds the theory of "contrecoup" in explaining a blow on the vault resulting in a fracture of the base. The theory is based on the assumption that the semi-fluid medium of the brain transmits shock waves to another site with resulting rupture. This presupposes that the skull is a sphere equally strong at all points filled with a non-compressible liquid. This is not completely true. A more likely explanation must be in the fracturing of weak points due to stress placed upon the entire skull from the blow. Basilar lesions may be classified according to their locations in the three fossae.

SYMPTOMATOLOGY OF THE ANTERIOR FOSSA

There are two cardinal signs of fracture in the anterior fossa; namely, hemorrhage and edema. Subconjunctival hemorrhage frequently appears at the outer canthus of the eye and progresses inward to the corneoscleral margin. In a severe case it may completely surround the cornea with sufficient chemosis to cut down the visual field. The maximum hemorrhage is usually reached in two days. The initial appearance is within a few hours of the injury. As would be supposed, the blood is bright red since there is a fresh extravasation.

Palpebral and peripalpebral hemorrhage is frequently seen in fracture of the anterior fossa. This differs from the subconjunctival type in that it starts at the inner canthus and progresses outward.

The extravasated blood may be entirely anterior to the suspensory ligament of the lid, indicating the fracture involves only the squama of the frontal bone. Since the cribriform plate is rarely spared in fractures in this fossa, subconjunctival, palpebral and peripalpebral hemorrhage are normally observed.

As one would expect, orbital hemorrhage may be quite extensive with actual protrusion of the globe. Severe proptosis, appearing, implies an injury to the cavernous sinus or internal carotid artery; whereas a moderate degree, occurring some hours after the injury, is usually due to orbital wall fracture which involves the ethmoidal vessels. If the proptosis occurs some days following trauma and slowly progresses, one must consider arteriovenous aneurysm, arising from a fracture near the sphenoid bone with a consequent communication between the cavernous sinus and the carotid artery. A number of writers have reported retinal hemorrhages associated with basilar fracture occurring on the same side as the injury. Fracture of the anterior fossa is almost always associated with bleeding from the nose and mouth, the mechanism being the tearing of the ethmoid vessels or a rupture of the cribriform plate. The blood flows from the nose and also into the pharynx where it is swallowed and frequently vomited.

The escape of cerebrospinal fluid indicates but one thing, that the fracture involves the cribriform plate with a laceration of the dura. It is important to differentiate between serous secretion and cerebrospinal fluid for upon this depends the treatment and prognosis. The tests are simple. Cerebrospinal fluid shows sugar, little or no albumen, and low chlorides; whereas, mucus has no sugar, high protein and chlorides. From the strictly clinical viewpoint, it may be considered cerebrospinal fluid if it occurs within 24 hours after injury, is colorless, profuse and continues for several days. However, laboratory examination should always be used to corroborate the clinical evidence.

NERVE INVOLVEMENT

The anterior fossa contains three nerve groups which may be affected by fracture. They are the optic, olfactory, and those passing through the superior orbital fissure—namely, the third, fourth, sixth and the ophthalmic division of the fifth.

The Optic Nerve. Blindness may be complete or partial and occurs as an immediate sequela. It is difficult to account for this condition when so many of the fractures occur away from the site of the optic foramina unless hemorrhage into the nerve sheath is the causative agent. Ruskin[3] suggests dislocation of the anterior clinoid processes and the consequent crushing of the nerve as the cause.

The Olfactory Nerve. Since a great majority of fractures of the anterior fossa involve the cribriform plate, the olfactory nerve is necessarily involved. The fine branches are always injured in this condition, and the bulb itself may be involved. It is difficult to estimate the extent of the loss of smell because of the usually serious condition of the patient and blood clots in the nose. Fortunately, partial recovery is the rule and permanent anosmia is rare.

The third, fourth, sixth and ophthalmic branches of the fifth. These nerves are rarely involved. When they are the disturbance is usually of a transitory nature from hemorrhage. The prognosis for return of function is good.

THE MIDDLE FOSSA

The cardinal finding of fracture of the middle fossa is hemorrhage from one or both ears, sometimes associated with escape of cerebrospinal fluid. Fractures of any consequence involving the middle fossa invariably extend into the temporal bone; in fact, clinically, the temporal bone is the middle fossa.

Fractures Involving the Temporal Bone: The temporal bone is involved in over one-third of all cases of basal skull fracture. Ballenger[4] gives labyrinthine involvement as 25%. There are three different types of fracture of the temporal bone, any or all of which may coexist:

1. longitudinal
2. transverse
3. rupture of the tip of the petrous portion

A longitudinal fracture, the most frequent type, starts at the mastoid tegmen and crosses the tegmen to the roof of the Eustachian tube. It is usually linear. However, if there are branches of the line of fracture, these extend to the external canal or the mastoid, rarely a middle ear fracture. As the tympanic membrane is ruptured, there is blood in the middle ear. The muscles, especially the tensor tympani, may be destroyed; sometimes there is dislocation of the incus. Inner ear hemorrhages occur only in severe cases, which are usually fatal. In about 30% of the cases of longitudinal fracture there is deafness of the conduction type. These fractures do not heal by bony union, and therefore present a constant danger of meningitis from subsequent otitis media.

Transverse fractures start at the jugular fossa, cross the pyramid to its anterior surface destroying the vestibule, and cause bleeding into the cochlea and canals. The middle ear is intact except when the fracture extends through its mesial wall with bleeding into the middle ear. Here again there is no bony union.

Symptoms of Fracture of the Temporal Bone:

1. Bleeding from one or both ears occurs in from 25 to 75% of all cases of basal skull fracture.

2. Cerebrospinal fluid escaping from the ear is less common than bleeding. When present it makes the diagnosis conclusive.

3. Deafness, partial or total, is a frequent accompaniment of fractures involving the temporal bone. It is of the nerve type unless the injury involves the middle ear only, in which case the conduction type of deafness will be present.

4. The finding of brain substance in the external auditory canal is rare, but when present is pathognomonic of basal skull fracture involving the temporal bone.

5. Loss of taste on the anterior two thirds of the tongue occurs when the chorda tympani nerve is injured.

6. Facial paralysis may occur if the seventh nerve is injured.

7. Tinnitus is usually present in incomplete destruction of the cochlear branch of the eighth nerve.

8. Vestibular disturbances such as vertigo, nausea, vomiting, spontaneous nystagmus toward the normal side, past-pointing etc. are present if the vestibular branch of the eighth nerve or the labyrinth itself is involved. The vestibular disturbances gradually and usually completely subside after several months or a year. With complete destruction of the labyrinth, all labyrinthine function tests give no response.

Diagnosis of Fractures of the Temporal Bone. In longitudinal fractures blood and cerebrospinal fluid in the external canal is positive evidence of a fracture with a tear of the dura. Redness of the superior wall of the canal with other signs is significant. Functional test shows deafness usually of the conduction type. The x-ray finding is reliable only if positive. Negative x-ray does not exclude fracture.

In transverse fractures the x-ray is more reliable. The drum membrane is usually intact, but the hearing is destroyed. The labyrinth does not respond to functional tests.

In fractures involving the petrous tip alone, the diagnosis is extremely difficult unless the sixth nerve is injured.

Prognosis of Temporal Bone Fractures. In longitudinal fractures the prognosis should be guarded since meningitis may occur at a later date due to extension of infection from an acute otitis media. The prognosis as to normal hearing is poor since the hearing usually deteriorates over the two to three months following injury.

In transverse fractures the prognosis is the same as in longitudinal fractures and for the same reason. The hearing is hopelessly impaired.

It should not be forgotten, particularly in cases of longitudinal fracture, that since no bony union follows, there is always a danger of meningitis in later years from extension of an otitis media.

Nerve Involvement: The fifth nerve's second divisions passes through the foramen ovale, and are only occasionally injured since they lie anterior to the petrosphenoidal suture which is rarely involved in a fracture.

The sixth nerve is more often involved, due to its course and the ease with which extravasated blood may cause pressure in the region of the petroclinoidal ligament (Dorello's canal). Fractures which involve the petrous tips are especially apt to affect the sixth nerve because of the intimate anatomic relationship.

The seventh and eighth nerves are particularly vulnerable because of their intrapetrous position and are frequently involved in fractures in this area. Their symptomatology was mentioned under the discussion of the temporal bone.

THE POSTERIOR FOSSA

Hemorrhage is a cardinal sign as in anterior and middle fossae fractures, but it differs in this fossa in that it becomes evident much later. Because of the nuchal muscles, the blood which is effused from the site of fracture has difficulty in rising to the surface and only makes its appearance in the form of ecchymosis 24 to 36 hours after injury. Occassionally, a peculiar ecchymotic patch is observed occurring anterior to the mastoid process and curving over the ear. This is said to result from blood following the course of the posterior auricular artery. Fractures involving the posterior fossa do not show the dramatic symptomatology of the other fossae, and frequently strictly clinical diagnosis is impossible.

Nerve Involvement: The ninth, tenth and eleventh nerves are rarely involved in this type of fracture unless the injury is most severe. A combined involvement of the tenth and twelfth may result in loss of speech and ability to swallow, paralysis of half the tongue, soft palate and pharyngeal constrictors. In the eleventh and twelfth nerve involvement; hypoglossal, sternomastoid, and trapezius muscle paralyses may be observed.

Roentgenograms: Some fractures of the base are not demonstrable by roentgenogram. Actually, no fracture of the base which does not include the calvarium is visualized in this manner. However, roentgenograms should be taken in all suspected fractures,

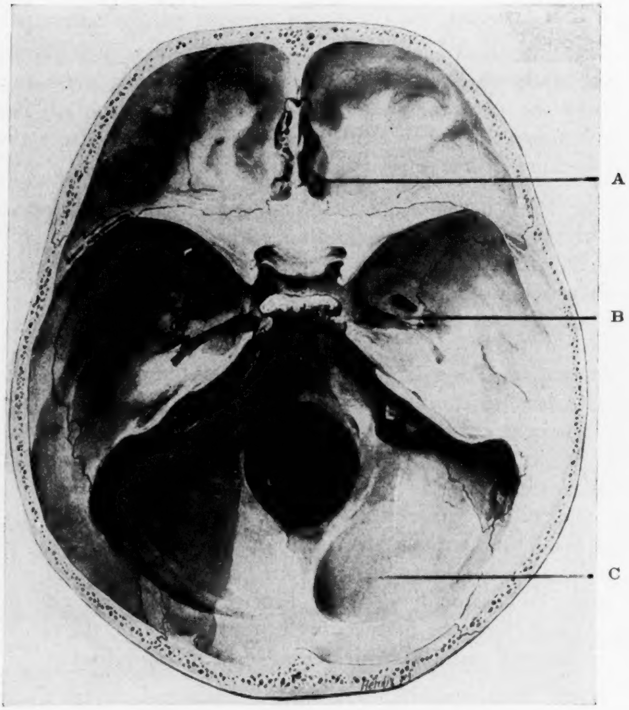

Fig. 1.—Weak areas of the base of skull. *A*. Anterior fossa; cribriform plate. *B*. Middle fossa; foramina. *C*. Posterior fossa; floor.

both because valuable information may be obtained and for protection from possible medico-legal difficulties. It is important to remember that the accurate interpretation of head films is difficult and requires expert opinion, since it is difficult to distinguish between a fracture line and a normal anatomical shadow. The grooves for the middle meningeal artery create very confusing shadows (Pancoast et al). These, as a rule, will be more hazy in their outline than those of fractures which are usually well defined, but sometimes a fracture line may simulate very closely the appearance of the middle meningeal artery groove. Occasionally, it is possible for a fracture to occur in a vessel groove, in which instance it is almost certain to be overlooked. Whether or not a fracture crosses a vessel groove should always be indicated in a roentgenographic report. Frequently, it is difficult to distinguish between a fracture line and the diploic canals, especially in the parietal region.

Treatment: Before any local treatment is instituted, shock, if present must be relieved. Fortunately, except in the most profound head injury, the amount of shock is not severe. The patient must be surveyed in his entirety for signs of severe injury to other portions of the body; such as hemothorax, ruptured viscera or injury to an extremity. A thorough neurological examination should be performed. This gives a base line from which progress may be noted. The neurological examination should note the state of consciousness; whether or not aphasia is present; size and regularity of the pupils; and whether or not there is motor weakness. The condition of the reflexes is important, as is the function of the cranial nerves.

Lumbar Puncture: The dynamics of the spinal fluid should be recorded and the fluid sent for laboratory examinations. The pressure, if elevated, should be reduced slowly to normal. While at variance with some of the older ideas, this has been found to be particularly effective in controlling advancing neurological signs. The spinal fluid pressure should be kept at normal fluid, but it always is present when there is a laceration of the brain. One may find the lumbar pressure normal or low. If normal it is obviously an excellent sign but if abnormally low it points to a severe injury, more so than when it is elevated. When the spinal fluid pressure is low, none but the absolute minimum amount should be removed.

Antibiotics and Chemotherapeutic Agents: In compound fractures, or where they are suspected, the antibiotic therapy must be pushed to adequate levels; penicillin in doses of at least 50,000 units every third hour and sulfadiazine sufficient to maintain eight to ten milligrams percent blood level.

Bed Rest: The value of early ambulation was demonstrated during the war. The criteria in skull fractures are normal spinal fluid pressure and disappearance of the initial neurological symptoms. It is necessary to adhere to the old empiric rule of at least two weeks or more flat in bed. Other general measures such as adequate supportive treatment and good nursing care are obviously essential in these cases.

Precaution: There are two conditions which need a more detailed method of treatment. The first is escape of blood and spinal fluid from the ear canal following temporal bone fracture. Here, indeed, is the place par excellence for "masterful inactivity" from the standpoint of local treatment. Sterile cotton placed in the concha to act as an absorbing agent is sufficient. The canal should not be packed, or an attempt made to instill medication, as this only tends to carry infection into the canal.

When there is an escape of spinal fluid from the ear canal, some have had the fear that the reduction of the spinal fluid pressure to normal would tend to suck back through the dura the contaminated fluid in the ear. This, however, is not true since it in no way constitutes negative pressure, but only a reduction of abnormal positive pressure.

The second condition is escape of spinal fluid from the nose following fracture of the cribriform plate; cerebrospinal rhinorrhea. Here as in the ear, packing or syringing is absolutely contraindicated. If the escape of fluid is in great volume, it is advisable to consider surgery, the operation of choice being a fascia transplant to close the dura at its point of laceration. Fortunately, in the great majority of cases the cerebrospinal fluid flow stops spontaneously.

CONCLUSIONS

1. Basilar skull fractures occur in the weakest structural areas.

2. The symptomatology of fractures of each fossa is distinct and individual.

3. Fractures involving the temporal bone do not heal by bony union and always present a potential danger of meningitis.

4. X-ray evidence of fracture as a diagnostic aid is valuable only if positive.

5. Keeping the cerebrospinal fluid pressure at normal levels hastens recovery from basilar skull fractures.

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LVI

THE RISK OF POLIOMYELITIS AFTER TONSILLECTOMY

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The existence of a relationship between poliomyelitis and tonsillectomy during the month preceding onset was first suggested by Sheppard[1] in 1911 and clearly shown by Aycock and Luther[2] in 1929. Since that time, numerous investigators have added to the growing literature on this topic, placing on record many instances of severe involvement that occurred within a few weeks after removal of tonsils. Undoubtedly many cases and even groups of cases remain unrecorded. Out of the many reports has come a growing realization that poliomyelitis occurring during the first month after tonsillectomy is more apt to be of the bulbar type than is infection occurring under other circumstances.

While few have disputed the predominance of bulbar infections in this group there have, however, been few specific data regarding the role of tonsillectomy as a factor in the development of a clinically recognizable poliomyelitis infection. Data have been lacking to determine the risk of developing poliomyelitis of any type by a recently tonsillectomized child as contrasted with one under otherwise identical circumstances who has not undergone recent tonsillectomy. Fischer, Stillerman and Marks[3] in a study of the 1941 outbreak in Toronto found the total attack rate among children 3-12 tonsillectomized during the preceding month to be twice as high as among those not undergoing such an operation and the bulbar attack rate ten times that of the control group. J. A. Anderson[4] observing the 1943 outbreak in Utah, found similar relationships.

That there has not been universal agreement with these findings and the conclusion that tonsillectomy during a poliomyelitis out-

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Aided by a Grant from The National Foundation for Infantile Paralysis.

break increases the risk of infection is shown by the report of Kinney[5] and the series of reports of Cunning.[6] The former in a study of 5 cases around Cleveland came to the conclusion that tonsillectomy did not increase the risk of recognizable infection as he found fewer cases occurring within a month after operation than might be expected as determined by the rate in the population under 19 years of age. One cannot agree with Kinney's methods of estimating the risk among tonsillectomized children as he makes no allowances for age differences and gives as much weight to tonsillectomies performed during the peak of the outbreak. Cunning in his annual reports has recorded cases of poliomyelitis occurring in various states within a month after tonsillectomy and has calculated the percentage of all poliomyelitis cases that fall in this group. While the recorded percentage is small—about 1.5%, this disregards the fact that in many parts of the country tonsillectomies are discouraged during poliomyelitis outbreaks. Similarly the results measure the importance of tonsillectomy as a factor in all poliomyelitis and do not express the added risk created by tonsillectomy.

The purpose of the current study is to record a series of at least 21 cases of poliomyelitis occurring within a month after tonsil removal and to attempt to measure the extent to which tonsillectomy adds to the risk of developing poliomyelitis. In 1946, Minnesota experienced an extensive outbreak of poliomyelitis, involving 2881 officially reported cases and 226 deaths. Detailed epidemiologic histories were obtained from 2709 cases. These 2709 include a small group of unquestioned poliomyelitis that was not officially recorded and omits another group of cases on which for one reason or another detailed histories could not be obtained. Included in this latter group were two known fatal bulbar infections in one family within a month after tonsillectomy, one of the deaths occurring in another state to which the family had traveled immediately after the first death. As full details are not available, these cases are omitted from further consideration in this report.

The 1946 outbreak in Minnesota reached its peak late in July in Minneapolis and early in August in the remainder of the state, though the actual peak varied somewhat in different parts of the state. The distribution of the survey cases by week is shown in Table I, showing that the spread of the infection was at its maximum late in July, declining rapidly in August and September.

In July, as it became apparent that a large number of cases of poliomyelitis could be expected, a warning was issued regarding the possible risk of tonsillectomy, and physicians and hospitals were urged to defer such operations, if possible. Many hospitals by formal action placed a ban on all but emergency cases; others discouraged operations. That the number of tonsillectomies actually declined is evident from Table II showing the number of such operations by months performed during the summers of 1945 and 1946. These data obtained by questionnaires addressed to the hospitals were received from 136 out of 186 general hospitals (excluding mental, tuberculosis and maternity). These 136 hospitals contain 92.44% of the 12,300 beds of this character in the state in 1946. There is no reason to think that the hospitals containing the remaining 7.56% of the beds had proportionately any fewer or more tonsillectomies. There is, of course, no way of knowing how many tonsillectomies may have been performed in the home or in a physician's office but knowledge of surgical practices in the state suggests that this number is so small as to be insignificant as compared with the large number in hospitals. It is apparent from comparison of Tables I and II that by the time the outbreak had reached its peak few tonsillectomies were being done.

The change in practice with respect to tonsillectomies was actually greater than is suggested by Table II. Of the 375 cases in August, 106 (28%) were performed in 4 hospitals and of the 469 in September, 88 (19%) were in these same 4 hospitals. Only 7.3% of the May tonsillectomies and 7.4% of the June operations were performed in these hospitals. It is of interest to note that the one case of poliomyelitis immediately following operation in August and the one in October were in the areas served by 2 of these 4 hospitals. Thus the hospitals where most of the tonsillectomies were performed almost discontinued operations. Analysis of the age distributions of the operations shows even further shift in practice. Table III based on a sample of five representative Twin City hospitals shows that operations on children decreased during July even more rapidly than did the total number of tonsillectomies and that in August and the first half of September essentially all the operations being performed were on adults.

The shift away from operations on children began about the middle of July and in some hospitals no operations on children were performed after July 20. It is thus apparent that the number of children who were tonsillectomized and thus could develop poliomyelitis within a month after operation was reduced to virtually zero between the middle of July and the middle of September.

Of the 2709 cases on which histories were obtained, 19 had had tonsillectomy or adenoidectomy during the previous month, 10 during the second preceding month and 8 during the third preceding month. Table IV shows the age, sex, date of operation and of onset, and type and outcome of each of these 19 cases. The breakdown by type of

TABLE I
POLIOMYELITIS CASES BY WEEK OF ONSET
MINNESOTA 1946

WEEK OF ONSET	NO. OF CASES
Jan. 1 - June 22	33
June 23-29	18
June 30 - July 6	64
July 7-13	96
July 14-20	186
July 21-27	264
July 28 - Aug. 3	319
Aug. 4-10	321
Aug. 11-17	263
Aug. 18-24	186
Aug. 25-31	174
Sept. 1-7	160
Sept. 8-14	132
Sept. 15-21	121
Sept. 22-28	93
Sept. 29 - Oct. 5	79
Oct. 6-12	52
Oct. 13-19	38
Oct. 20-26	28
Oct. 27 - Nov. 2	22
Nov. 3-9	14
Nov. 10-16	17
Nov. 17-23	8
Nov. 24-30	7
Dec. 1-7	3
Dec. 8-31	11
Total	2709

TABLE II
TONSILLECTOMIES IN MINNESOTA HOSPITALS
1945 AND 1946

	1945	1946	
		REPORTED*	ESTIMATED TOTAL
May	1696	1689	1827
June	4296	3989	4315
July	4530	2722	2945
August	4476	375	406
September	1804	469	507

*Reports from 136 of 186 general hospitals representing 92.4% of all available hospital beds.

infection is shown in Table V. It is apparent that not only were there more cases in the group developing infection within a month after operation but also that 12 of the 19 (63.2%) were bulbar as contrasted with only 4 bulbar out of 18 cases (22.2%) in the 2nd and 3rd months. During the entire outbreak 20.0% of all patients and 11% of those under 12 had bulbar infection. These data are in keeping with those previously recorded in showing an undue concentration of bulbar type among those most recently tonsillectomized.

Tables VI and VII further summarize the data of Table IV. From Table VI it will be noted that all except two of the cases were between 3 and 7 years of age. One of these exceptions was an 11 year old girl who developed a mild paralytic infection two days after an adenoidectomy. The incubation period was so short that one may safely discard the case as having no probable causal relationship between the operation and subsequent infection. The other was an 11 year old boy who had his operation late in August on the decline of the epidemic wave when very few tonsillectomies were being performed and almost none on children. He developed a nonparalytic poliomyelitis 8 days later, the only nonparalytic infection in this series.

Table VII summarizes the data regarding date of operation and onset. With two exceptions all of the 19 had had their operations between June 12 and July 25 and onsets between June 23 and August 7. The exact dates are shown in Table IV; they are summarized in Table VII according to the week in which they occurred. That only two of the 19 should have had their operations after July 25 is not surprising in view of the general discontinuance of tonsillectomies in children during the outbreak as noted above.

Table V, already referred to, further summarizes the data with respect to incubation period and type of infection. Of the 19 cases, 12 were bulbar, 6 paralytic and one nonparalytic but these 19 include cases which on the basis of incubation periods seem probably unrelated to the previous tonsillectomy. If one accepts the incubation period of poliomyelitis as probably between 7 and 20 days, it is apparent that 12 of the 15 (80%) cases with incubation periods in this range developed bulbar infection whereas none of the balance had bulbar type. The three fatal cases had incubation periods of 12, 13 and 14 days respectively. It would appear from this table that not only is there a concentration of cases about 7-20 days after operation, but also that these cases tend to be of the more severe bulbar type of infection. These findings are in keeping with those of other studies.

TABLE III
AGE DISTRIBUTION OF TONSILLECTOMIES BY DATE OF OPERATION—FIVE TWIN CITY HOSPITALS, 1946

	AGE OF PATIENT			
DATE	UNDER 12	12-19	20	TOTAL
6/1-15	172	24	59	255
6/16-30	193	54	50	297
7/1-15	140	19	41	200
7/16-31	41	11	32	84
8/1-15	0	1	12	13
8/16-31	0	2	9	11
9/1-15	0	4	8	12
9/16-30	30	10	34	74

It is of course possible, however, that while tonsillectomy may be a factor in conditioning a bulbar response if infection is developing, it may not increase the risk of contracting recognizable poliomyelitis. That the risk is greater is suggested, however, by the much larger number of patients who contracted poliomyelitis during the first month posttonsillectomy than during the second or third month after operation. In this study there were 19 cases in the first group and 18 in the other two combined. The large number in the one-month group is caused by a concentration of bulbar cases occurring 7-15 days after operation. This concentration during the apparent range of the incubation period is in itself strongly suggestive of a causal relationship.

A better measure of the risk involves comparison of the attack rate among all tonsillectomized children and that of children of comparable age group during the same period of time. Rates based on all children developing poliomyelitis regardless of age and irrespective of the period of the outbreak are meaningless since the risks of developing the infection vary so greatly at different ages and stages of the epidemic cycle. The only truly valid comparisons are those based on groups of equal age and periods of exposure.

Of the 19 children who developed poliomyelitis within one month after tonsillectomy, one, a mild paralytic case of 11, had her operation (an adenoidectomy only) on July 16 and developed poliomyelitis on July 18. This case can reasonably be discarded as probably not causally related due to the interval of only two days between operation and onset. Another case, a boy of 11 with nonparalytic involvement had his operation on August 23 and his onset August 31 while a third, a fatal bulbar case of 6, had his operation on October 3 and became ill on October 15.

As already shown in Table III, tonsillectomies in children had essentially ceased by August 1. A single case in August and one in October occurring in such small groups could have been so much a matter of mere chance (even though the risk might be numerically increased, any rate based on a single case is obviously unreliable) that it seems best to omit these also from consideration. Thus three cases are omitted; inclusion of any or all of these would have increased the apparent risk of tonsillectomy. There remain 16 cases for further consideration.

All of these 16 were between the ages of 3 and 7 inclusive. Their operations were performed between June 12 and July 25 (weeks ending June 29 and August 10 respectively). During this same period, 508 children ages 3-7 inclusive developed poliomyelitis in Minnesota, 491 of whom had not had tonsillectomy or adenoidectomy during the preceding month.

To determine the risk of poliomyelitis following tonsillectomy and adenoidectomy it is necessary to know the number of such operations performed on children of this age during this same period of time. Table II shows that in June and July, 1946, there had been 3989 and 2722 tonsillectomies respectively. If it is assumed that the hospitals that did not reply to the questionnaire had had the same ratio of such operations per bed, this would have meant an estimated 4315 tonsillectomies in June and 2945 in July, or a total of 7260.

The exact age and date of these operations is not known. A sample of five hospitals in the Twin Cities in which 836 operations were performed in these two months showed that 51.0% of the operations were on children 3-7 inclusive and that 53.8% of the June operations had been done during the period after June 15. If this is a representative sample of the total state (and there is no reason to believe that the distribution by age or date in June would be different elsewhere) one may assume 53.8% of the 4315 in June were in the latter half, making a total of 5266 performed between June 16 and July 31. Of these, 51.0% or 2686 were aged 3-7 inclusive. Of this group of 2686, there were 16 who developed poliomyelitis or a ratio of 1 to 168 operations (0.60%). The bulbar attack rate was 1 to 224 (0.45%).

TABLE IV
POLIOMYELITIS CASES DEVELOPING WITHIN ONE MONTH AFTER TONSILLECTOMY — MINNESOTA 1946

AGE	SEX	DATE OF T & A	DATE OF ONSET	INTERVAL IN DAYS	TYPE OF CASES	OUTCOME
3	F	6/12	7/11	29	P	P
3	F	6/16	6/23	7	B	P
7	F	6/25	7/14	19	B	P
6	F	6/29	7/12	13	B	P
3	F	6/30	7/22	22	P	NP
6	M	7/5	7/13	8	B	P
6	M	7/6	7/18	12	B	NP
4	M	7/9	7/22	13	B	P
7	M	7/11	8/3	23	P	NP
7	M	7/12	7/29	17	B	D
11	F	7/16	7/18	2	P	NP
5	F	7/16	7/31	15	P	NP
5	M	7/16	8/1	16	B	P
5	F	7/22	7/31	9	B	NP
4	M	7/23	8/2	10	P	NP
7	M	7/24	8/3	10	B	NP
5	M	7/25	8/7	13	B	D
11	M	8/23	8/31	8	NP	NP
6	M	10/3	10/15	12	B	D

D—Death
B—Bulbar case
P—Paralyzed case
NP—Non-Paralyzed case. When in column headed "Outcome" it refers to cases without disabling paralysis tho residual weakness may have been present.

To determine the significance of this attack rate it is necessary to know the attack rate during the same period for children of the same age who did not have a tonsillectomy. It has been pointed out above that 491 children who had not had tonsillectomy developed poliomyelitis during the weeks ending June 29 to August 10 inclusive, the same weeks during which these 16 tonsillectomized children had their onsets. The child population ages 3-7 inclusive of Minnesota in 1946 is obviously unknown. Extrapolation from the 1930 and 1940 census data yields an estimate of 222,894. This is obviously too low an estimate as the birth rates since 1940 have been elevated. Another estimate of the size of this age group can be made from numbers of births in the appropriate years with subtraction of the known subsequent deaths. This latter estimate assumes either no gain or loss by migration, or equality of gain and loss. On this basis the number of children ages 3-7 inclusive is estimated as 264,036. A third estimate may be made by taking the average of those two figures. From these estimated populations it is necessary to subtract 2686 as the number who underwent tonsillectomy during the period June 16 to July 31. The number of control children can thus be estimated as 220,208, or 261,350, or 240,779 respectively. Of these, 491 developed poliomyelitis during the period in question. This makes an attack rate of 1 to 448 (0.22%) or 1 to 532 (0.19%) or 1 to 490 (0.20%) respectively. Whichever estimate is accepted makes little difference. In any case the attack rate for the group whose tonsils were removed during the preceding month is three times or more greater than among those undergoing no such operation. Of the 491 who developed poliomyelitis, 96 were bulbar infections. This makes a bulbar attack rate of 1 to 2508 (0.04%) a rate only one-eleventh that of the tonsillectomized group.

The above comparisons between the attack rates of recently tonsillectomized children and those not tonsillectomized are obviously conservative estimates. One case of poliomyelitis following tonsillectomy has been omitted as the incubation period was too short to suggest causal relationship. Two fatal bulbar cases were omitted as the family moved out of the state and full details were not obtainable. They were both in the age groups and incubation periods considered here, and their tonsillectomies are included in the total operations. The single case tonsillectomized in August has been further omitted as so few tonsillectomies were done in that month that it seemed unfair to base any conclusions on a single case. Another case has been omitted that occurred in October when few tonsillectomies were being done and the risk in the control group had further declined. Exclusion of all of these seems justifiable to

TABLE V

TYPE AND SEVERITY OF POLIOMYELITIS IN RELATION TO TIME INTERVAL BETWEEN TONSILLECTOMY AND ONSET

MINNESOTA 1946

INTERVAL (DAYS)	TOTAL	BULBAR	PARALYTIC	NON-PARALYTIC	DIED
0-6	1	--	1	--	--
7-10	6	4	1	1	--
11-15	6	5	1	--	3
16-20	3	3	--	--	--
21-31	3	--	3	--	--
	19	12	6	1	3
1-2 months	10	2	6	2	--
2-3 months	8	2	2	4	1

TABLE VI

POLIOMYELITIS FOLLOWING TONSILLECTOMY

AGE DISTRIBUTION OF CASES OCCURRING WITHIN 1ST MONTH POST-TONSILLECTOMY

MINNESOTA 1946

AGE	BULBAR	PARALYTIC	NON-PARALYTIC	DIED
3	1	2	--	--
4	1	1	--	--
5	3	1	--	1
6	4	--	--	1
7	3	1	--	1
11	--	1	1	--
	12	6	1	3

TABLE VII
POLIOMYELITIS FOLLOWING TONSILLECTOMY WEEKS OF OPERATION AND ONSET IN CASES DURING 1ST MONTH POST-TONSILLECTOMY

WEEK ENDING	OPERATION	ONSET
June 15	1	--
22	1	--
29	2	1
July 6	3	--
13	3	3
20	3	3
27	4	2
Aug. 3	--	7
10	--	1
17	--	--
24	1	--
31	--	1
Oct. 3	1	--
15	--	1
	19	19

TABLE VIII
POLIOMYELITIS ATTACK RATES IN TONSILLECTOMIZED AND IN NON-TONSILLECTOMIZED CHILDREN MINNESOTA, JUNE 15 - JULY 31, 1946

	TONSILLECTOMIZED**	NON-TONSILLECTOMIZED***
Population 3-7	2686	240,779*
Cases of Poliomyelitis	16	491
Attack Ratio	1/168 (0.60%)	1/490 (0.20%)
Bulbar Cases	12	96
Attack Ratio	1/224 (0.45%)	1/2508 (0.04%)

*Average of two estimates. See text for explanation of methods of estimation.

**Tonsillectomized during weeks ending 6/15/46 thru 7/31/46; developed poliomyelitis during weeks ending 6/29/46 thru 8/10/46.

***Developed poliomyelitis during weeks ending 6/29/46 thru 8/10/46.

achieve accurate statistical comparability. Had any or all of them been included, the rate in the tonsillectomized group would have been higher and the differences in the two groups greater. The estimate of a threefold risk of infection and elevenfold risk of bulbar infection as a result of tonsillectomy seems therefore to be a conservative one. It is recognized, of course, that the number of cases is small, yet the difference is statistically significant.

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CONCLUSIONS

1. During the 1946 outbreak of poliomyelitis in Minnesota, 21 cases occurred within one month following tonsillectomy. Details of 19 of these are presented. Five (3 of the 19) were fatal.

2. Twelve of the 19 (63.2%) were bulbar as contrasted with only 4 bulbar out of 18 cases (22.2%) who had tonsils removed in the 2nd and 3rd months preceding onset.

3. There was a concentration of bulbar cases whose onset after operation was within the apparent usual range of incubation periods.

4. The risk of developing poliomyelitis was at least three times as great among those undergoing tonsillectomy as among a comparable group not undergoing this operation and the risk of bulbar infection was 11 times as great.

The authors are indebted to the many hospitals in Minnesota which furnished data for this study and particularly to the Fairview, St. Mary's, St. Barnabas and Swedish Hospitals in Minneapolis and to the Miller Hospital in St. Paul which permitted more detailed study of their records.

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LVII

MULTIPLE PRIMARY MALIGNANT TUMORS

REPORT OF THREE CASES ENCOUNTERED ON AN OTOLARYNGOLOGICAL SERVICE

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Multiple primary malignancies are not rare. A review of cases reported by various American investigators since 1932 shows an average incidence of 2.6 per cent (Table I). While the incidence of multiple malignancies has varied little, the interest in this condition has apparently increased. The Quarterly Cumulative Index Medicus in 1937 listed twelve articles on the subject but in 1948 the Index listed twenty-one articles for the first half year.

The literature of Otolaryngology shows a paucity of reference to the subject of multiple primary neoplasms. This is in spite of the fact that Spain[12] in reporting a series of cases of multiple malignant tumors from Bellevue Hospital in 1949, found in 20 cases that the bronchus was the site of one of the primary tumors in 7 cases and the esophagus in 2 cases. Drooker[4] in 1937 reported a case of three primary malignancies encountered in a patient on a Nose and Throat Service. Tolczynski[14] in 1947 reported a case of multiple primary carcinoma of the larynx. Others have reported multiple malignancies of the oral cavity. Warren and Gates[15] called attention to multiple cancers of the pharynx and considered syphilitic leukoplakia a factor. However, it is likely that multiple tumors occurring in a single organ should be considered as tumors of multicentric origin. Such tumors are common in the skin, bladder and gastro-intestinal tract.

Billroth[1] in 1860 first reported multiple primary malignancies and established criteria for such a diagnosis. These criteria have

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"Reviewed in the Veterans Administration and published with the approval of the Chief Medical Director. The statements and conclusions published by the author are the result of his own study and do not necessarily reflect the opinion or policy of the Veterans Administration."

TABLE I.

AUTHOR	YEAR	POST-MORTEMS WITH CARCINOMATA	MULTIPLE CARCINOMAS	PER CENT
Warren & Gates	1932	1078	40	3.7
Hurt & Broders	1933	2124	71	3.34
Bugher	1934	983	28	3.1
Schreiner & Wehr	1934	11212	307	2.7
Burke	1936	583	46	7.8
Kirschbaum & Shirley	1938	10870	25	1.77
Stalker, Phillip & Pemberton	1939	2500	113	4.5
Peller	1941	5876	270	3.9
Hellendall	1943	685	30	4.3
Spain	1949	690	22	3.2
TOTAL		36601	952	2.6

been found too rigid and have been replaced by the following: (1) The presence of neoplasms at separate sites; (2) Each neoplasm must have an independent histologic appearance; (3) The signs of malignancy must be unmistakable; for example, infiltration into the surrounding tissue. The possibility of one of the lesions being a metastatic growth should be excluded. The presence of metastases is no longer considered necessary as one of the criteria of multiple malignancies. The tumors may be synchronous, occurring simultaneously or metachronous, occurring at different times.

Hill[6] believed that multiple neoplasms could be explained by the laws of chance. Warren and Gates,[15] however, who made an exhaustive study of the subject in 1932, believe that it is explained by a definite susceptibility to cancer present in certain individuals.

White,[16] in 1941, considered the predisposition of certain individuals to develop multiple neoplasms as due to the interaction of a functionally mature gene-bearing unit for cancer inheritance with one for localization plus necessary stimuli from the environment.

Spain[12] believed that the high incidence of multiple malignancies supported the mutation theory of the origin of cancer.

As many as four primary malignancies in different organs have been reported. Goldstein and Rubin[5] in reporting one such case found ten other cases in the literature up to 1948.

The following three cases of multiple primary malignancies were encountered on the Nose and Throat Service of the Baker Veterans Administration Hospital within a period of fifteen months.

CASE 1.—W. H. aged 62, white male, was first seen on October 4, 1948, because of profuse nasal discharge. Examination at this time revealed a nasal mucosa that was pale and boggy suggestive of allergy. On the left anterior faucial pillar there was an area of elevated, white-appearing tissue which bled easily on manipulation. Biopsy at this time showed squamous epithelium with acanthosis and parakeratosis but with no evidence of malignancy. The patient was again seen on December 7, 1948, and another biopsy made. The cells in the section were found to be hyperchromatic with large, irregular nuclei having many mitotic figures. There was some penetration into the supporting stroma. The diagnosis was squamous carcinoma, fairly anaplastic.

The patient was admitted to the hospital for operation on January 1, 1949. His past history revealed that he had a left pneumonectomy performed at the V. A. Hospital, Aspinwall, Pennsylvania, on April 14, 1947, for squamous carcinoma grade III of the left bronchus. Following operation, the patient had deep x-ray therapy to the bronchial stump. Examination of the removed lung showed a granular area of mucosa in the left main bronchus extending to the bronchus of the upper lobe and the bronchus of the superior portion of the lower lobe. Section from the tumor showed scattered islands of large pleomorphic, hyperchromatic cells occasionally surrounding epithelial pearls. Intracellular bridges were readily identified. The diagnosis was carcinoma, squamous cell, grade II. Following surgery, the patient slowly convalesced and was discharged on March 29, 1948. Additional findings in the past history were tonsillectomy in 1936 and treatment for syphilis in 1928. There was no family history of cancer.

Physical examination at time of admission to Baker Hospital showed a fairly well developed and nourished male patient. There was an indurated area of mucosa over the left anterior faucial pillar and a tonsil tag in the left fossa which bled easily. There was a thoracic scar of the pneumonectomy. The right lung was clear to ausculation. The other physical findings were normal.

Laboratory and x-ray findings were as follows: Blood Kahn was positive (32 Kahn units). The white blood count numbered 6100. The hemoglobin was 12.5 gms. Urinalysis was normal. Chest x-ray showed a homogeneous density of the left lung field with a shifting of the trachea and mediastinal structures to the left. The left diaphragm was elevated.

On January 4, 1949, by means of surgical diathermy, the left anterior and posterior faucial pillars, the remaining tonsil and part of the lateral margin of the tongue were removed in one mass. Section from this showed the epithelium invading the underlying tissue in large, rounded masses composed of irregularly disposed epithelial cells varying considerably in size and shape and containing numerous mitotic figures. Diagnosis was squamous cell carcinoma.

The patient was discharged from the hospital to the Domiciliary Section where he has been kept under observation. Up until the present time there has been no evidence of recurrence nor metastases from the oral or the pulmonary tumor. Diagnoses were 1. Squamous cell carcinoma of bronchus, operated at Aspinwall Hospital, April, 1947. 2. Squamous cell carcinoma of pharynx, operated at Baker Hospital, January, 1949.

CASE 2.—W. L. aged 52, colored male, was first seen on November 8, 1948, because of a painful ulceration in the floor of the mouth.

The patient had been admitted to the medical service on October 29, 1948. His present illness dated back four years when he first began to have symptoms of "tightness" in his epigastrium and cramp-like pains in his legs and abdomen. He was examined and treated at two Philadelphia hospitals but had increasing weakness and dizziness. In July, 1948, he was admitted to the U. S. Naval Hospital in Philadelphia because of a weight loss of 32 lbs. From here, he was transferred to the Baker V. A. Center. On admission, his chief complaints were choking sensations in the throat and chest, pain in the floor of the mouth, weakness and abdominal pains. He also complained of impaired vision.

His general health had been good up until the present illness. Eight years ago he had sustained a fractured skull when struck in the occipital region with a brick. There was no history of cancer in his family.

Physical examination revealed a rather emaciated, male, negro patient. The left pupil was dilated and reacted sluggishly to light. There was some hazziness of the lens in the left eye. There was a scar and depression over the occiput. In the floor of the mouth on the left, near the orifice of the submaxillary duct, there was an ulceration about 0.5 cm in diameter with indurated edges. The left submaxillary gland was tender on palpation. The remainder of the physical examination showed no pertinent abnormality. The blood pressure was 110/80. The x-ray and laboratory data were as follows:

red blood count 4.02 million, hemoglobin 12.5 gms, white blood count 7,100 with a normal differential. The blood Kahn test was negative. Urinalysis was normal. Gastric analysis showed a histamine-fast achlorhydria. Chest x-ray showed normal heart and lung fields. An upper G. I. series showed considerable elongation and irregularity of the lower portion of the esophagus associated with some delay in emptying. There was a marked filling defect of the mesial aspect of the cardia.

On November 8, 1948, an excision biopsy was performed on the ulceration in the floor of the mouth. The microscopic examination of this tissue was reported as follows: There is a densely packed mass of hyperchromatic cells varying in size and shape. The nuclei also vary in size and shape. Some are large and irregular with heavy chromatin network. There is a linear and plexiform arrangement of cells with abundant mucinous inter-cellular stroma. Some cells are fusiform and vacuolated. In the margin there is a dense infiltrate of round and plasma cells. There is some normal-appearing salivary gland tissue present. Diagnosis: Pleomorphic adenoma of salivary gland with malignant change.

On November 26, 1948, an esophagoscopy was performed. On reaching the level of the diaphragm, the lumen was found to be narrowed by a hard, firm growth underlying a normal-appearing mucosa. A No. 16 French dilator was passed into the stomach and upon withdrawing this, some tissue fragments were present on the dilator. These were sent to the laboratory for examination. The report was as follows: "The esophageal mucosa is very acanthotic and there are prolongations of the deep layers into the submucosa. These are composed of masses of cells which are extremely anaplastic and hyperchromatic. The nuclei vary in size and shape and there are numerous mitotic figures. In one area there is a large mass of hyperchromatic cells without particular architectural arrangement. Many areas of this mass are undergoing necrosis. Diagnosis: Squamous carcinoma of the esophagus, extremely anaplastic."

The patient refused any surgery for the lesion in his esophagus and stomach. His course was progressively down hill and he expired February 19, 1949. Post-mortem examination was reported as follows:

"There was an ulceration about 1 cm in diameter in the floor of the mouth. Microscopic section of this area revealed normal salivary gland with the margin of the ulcer lined with fibrin and many round cells and polymorphonuclear leukocytes. No neoplastic tissue was round.

The liver could not be separated from the stomach. A large, yellow-gray tumor mass was present in the liver continuous with a mass in the cardiac portion of the stomach which encircled the esophageal orifice. Many smaller nodules were scattered throughout the liver. Microscopic section through this mass showed the hepatic tissue invaded by a mass of anaplastic cells with numerous mitotic figures.

Examination of the gastro-intestinal tract showed an ulcerating, fungating mass extending from the lower portion of the esophagus into the cardiac portion of the stomach and into the liver. The mass was about 8 cm in diameter. Section of this tissue showed stomach wall invaded and replaced by masses of cells varying in size and shape and having hyperchromatic nuclei with numerous mitotic figures. There were many areas of necrosis. Multi-nucleated giant cells were present. In most places the tumor was undifferentiated."

The remainder of the post-mortem findings were not significant.

The final post-mortem diagnoses were:

1. Squamous carcinoma of the esophagus with extension into the cardiac portion of the stomach and liver.

2. Pleomorphic adenoma of salivary gland with malignant change verified by microscopic examination of biopsy specimen.

3. Chronic pericarditis, healed.

Case 3.—C. T., aged 58, white male, was admitted to the Ear, Nose and Throat Service January 24, 1950, complaining of some difficulty in swallowing which he dated back only seven weeks. He also had some intermittent huskiness to his voice. His general health had been good up until 1948 when he began having dyspnea on exertion and swelling of both ankles. He had been hospitalized in this hospital for one month in April 1949. Diagnosis made at that time was arteriosclerotic heart disease. There was no history of cancer in the family.

Physical examination at the time of admission showed a fairly well developed white male with slight evidence of malnutrition. The left vocal cord was thickened and nodular in appearance and there were several white plaques on the surface. The cervical lymph nodes were enlarged on the left side. The apical was 100 compared with a radial pulse of 82. The examination otherwise was not remarkable.

Laboratory and x-ray findings were as follows: white blood count were 15,000 with 80 per cent polymorphonuclears. Red blood count were 3.2 million. Hemoglobin was 10.5 gms. The Kahn test was negative. Urinalysis was normal. Chest x-ray showed the lung fields to be clear. The heart appeared to be enlarged. There was a soft tissue shadow in the posterior mediastinum just behind the heart. Fluoroscopy and radiography of the esophagus with barium showed a marked irregular filling defect with narrowing of the esophagus from the level of D-6 to D-12. The stomach was distended due to some obstruction at the pylorus, either cicatrization of an old ulcer or possibly spasm.

On January 26 under direct laryngoscopy, a piece of tissue was removed from the left vocal cord. The report on this section was as follows: "The laryngeal epithelium is hyperplastic and hyperkeratotic. Clumps and nests of cells have broken through the basal layer and are found in the underlying tissue. The cell nuclei have numerous mitotic figures and less of polarity. There is much evidence of abnormal keratinization in the invading cell masses and no definite arrangement of their component cells. Diagnosis was squamous cell carcinoma, Grade II."

On January 31 an esophagoscopy was performed and a fungating mass constricting the lumen was found 29 cm from the upper teeth. A piece of tissue was removed and sent to the laboratory. The following report was received: "The section shows keratinized debris and masses of tumor composed of fairly well differentiated squamous epithelial cells. These vary in size and shape and show abnormal keratinization as well as numerous mitoses and tumor giant cells." Diagnosis was squamous cell carcinoma, Grade II.

The patient was seen by the thoracic surgeon who felt that resection of the esophagus was out of the question because of his cardiac condition and the fact that an obstructive lesion of the pylorus would necessitate a partial resection of the stomach in addition. It was also decided not to treat the lesion in the larynx. Jejunostomy was performed on February 21, 1950.

On May 1, 1950, the patient's temperature became elevated and he complained of pain in the left chest. X-ray showed evidence of pneumonitis of the lower lobe of the left lung and a pleural effusion on the same side. Antibiotic therapy was instituted but the patient expired on May 7, 1950. Post-mortem examination was performed.

The final post-mortem diagnoses were:

1. Squamous cell carcinoma, grade II, of larynx.
2. Squamous cell carcinoma, grade II, of esophagus.
3. Arteriosclerotic heart disease.
4. Pneumonitis, acute, suppurative, lower lobe of left lung.
5. Pleuritis, acute, suppurative with effusion, left.

SUMMARY

Three cases of multiple primary malignancies are reported. The neoplasms in one case were metachronous and in two cases were synchronous. The diagnosis in the two synchronous cases was substantiated by post-mortem examination. The patient with the metachronous neoplasms is still alive and well with no sign of recurrence of either tumor.

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LVIII

THE EFFECTS OF DRAMAMINE UPON COCHLEAR FUNCTION AND THE VESTIBULAR RESPONSES TO TURNING IN NORMAL SUBJECTS

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The value of dramamine (B-dimethylaminoethyl benzohydryl ether 8-chlorotheophyllinate) in the prevention and treatment of motion sickness was first pointed out by Gay and Carliner.[1] This drug also occasionally relieves the vertigo in prolonged attacks of Meniere's symptom complex. Campbell[2] has found it effective in modifying the vertigo and spontaneous nystagmus resulting from the reactive serous labyrinthitis which usually immediately follows the fenestration operation for otosclerosis.

Schiff[3] has shown in rabbits that dramamine has a repressive effect upon the central portion of the vestibular mechanism at the nuclear level. However, the pharmacologic action of the drug has not been fully explained.

We have studied the effects of dramamine, dramamine and morphine, and morphine alone upon the vestibular responses of normal medical students. We have attempted to explain the pharmacologic mechanism of dramamine as well as its locus of action.

Two of us[4] have shown elsewhere that the results of turning tests of vestibular function performed on the same normal individual one week apart vary slightly in a purely random fashion. If dramamine should influence the vestibular responses to an extent demonstrable by this method, an alteration from this random variation might be anticipated.

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METHOD

Eleven normal male medical students (ages 22 to 29) served as subjects. Two examinations, one week apart, were performed on each individual. Two hours and again one hour before one examination, 100 mg of dramamine were taken by mouth. Before another examination a placebo was similarly ingested. Each subject thus served as his own control. The examinations were made 45 minutes to one hour after lunch. After resting quietly in a chair for 15 minutes each subject, with his eyes closed, his head so fixed that an imaginary line between the external canthus of his eye and his tragus was in a horizontal plane, was turned in a standard Barany chair 10 times in 20±0.25 seconds. Following rotation, each subject looked without visual fixation straight forward into the distance and indicated the instant at which all sensation of rotation ceased. This was called the duration of post-rotational vertigo. The nystagmus was observed concomitantly, and its precise duration as noted by the unaided eye of the investigator recorded (the same observer was employed throughout the study). Three minutes after the cessation of nystagmus and vertigo, the subject was turned in a similar manner in the opposite direction. A single examination thus consisted of a rotation as just described, followed three minutes after the disappearance of nystagmus and vertigo by a repetition of the turning in the opposite direction. Audiograms were made after each vestibular examination in a sound-conditioned room by Miss Marian Knorr. The same audiometer was employed throughout these studies.

RESULTS

Following the administration of dramamine, there was no statistically significant alteration in the duration of the responses to turning for the nystagmus and the vertigo as compared with the results after a placebo (Table I). All of the subjects had normal hearing following the placebo. After dramamine, two of the eleven subjects (who were examined on different days) showed a loss of hearing which increased at and above 1024 double vibrations per second (Fig. 1). The audiometric curves were almost identical in both subjects. Repetition of the audiograms several days later revealed normal hearing. Three weeks later audiograms were repeated on one of these two subjects without, and again with, dramamine. The hearing was then found to be normal.

In another experiment, reported in detail elsewhere,[5] six of these same subjects received 100 mg of dramamine orally, followed one hour later by 15 mg of morphine sulfate subcutaneously. One

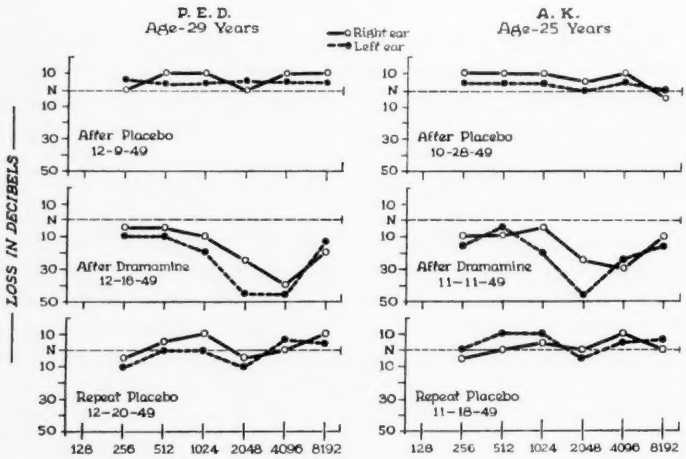

Fig. 1.—Audiograms showing the effect of dramamine upon cochlear function in two normal subjects.

hour later 100 mg of dramamine were again taken by mouth. One hour after this, that is two hours after the administration of the morphine, the turning test was performed as described above. A similar trial a week later was performed with morphine, but with a placebo substituted for the dramamine.

RESULTS

There was no significant difference in the vestibular responses between the morphine-placebo and the morphine-dramamine combinations. However, the difference in symptoms between the two trials was marked. With morphine plus dramamine none of the subjects had any complaint of nausea, nor was there any emesis following vestibular stimulation. They uniformly stated that they felt excellent. Furthermore, they gave little objective evidence of sleepiness or other difficulty. With the morphine-placebo combination, five had severe nausea and four vomited violently (Table II).

DISCUSSION

Schiff[3] has shown that dramamine prevents, in rabbits, the "adversive" syndrome of Himwich. Himwich et al[6] have shown that when diisopropylfluorophosphate (D.F.P.) is injected into one carotid artery of a rabbit, cat, dog, monkey, or guinea-pig, the "adversive" syndrome results. This consists of the turning of the animal's head with circling movements of the animal in the opposite direction to the injected carotid artery, a nystagmus in the same direction, and a contraction of the pupil of the homolateral eye. They have shown that D.F.P. causes a marked decrease in brain cholinesterase as a result of which the activity of acetylcholine is increased at the neuronic synapses. This increased acetylcholine activity produces an increase in the flow of nerve impulses through the chain of vestibular neurons on the injected side. This is in effect similar to an irritative lesion of the vestibular end organ or of the vestibular nuclei in the central nervous system. Spiegel[3] has experimentally shown that D.F.P. has no effect upon the vestibular end organ. One must, therefore, infer that the simulated irritative phenomena producing the "adversive" syndrome emanate at the vestibular nuclear level.

There is thus an apparent discrepancy between Schiff's[3] experiment, in which he concluded that dramamine has a repressive effect upon the vestibular mechanism at the nuclear level, and our experiments, in which we found no effect of dramamine upon vestibular function. This discrepancy might be reconciled on the basis of the theory of the chemical mediation of nerve impulses through a series of neurons.[7] In practically all of the central nervous system, including the parasympathetic and the preganglionic neurons of the sympathetic nervous system, the transmission of nerve impulses from one neuron to the next is dependent upon the presence of the chemical agent acetylcholine at each synapse. All nerves which depend upon acetylcholine for the transmission of nerve impulses are designated as cholinergic nerves. The postganglionic sympathetic nerve fibers depend upon epinephrin for the transmission of nerve impulses. These nerves are designated as adrenergic nerves. The vestibular mechanism and its communicating nerve centers in the brain, such as the emetic and vagus nerve centers, belong to the cholinergic system.[8] We shall therefore confine our remarks to the cholinergic mechanism.

Cholinesterase, an enzyme which is present in great abundance in brain tissue, and in all tissues where acetylcholine is liberated by nerve impulses, rapidly hydrolyzes acetylcholine into acetic acid and choline. As a result, the effect of acetylcholine is brief in duration. An increase in the quantity or the activity of acetylcholine at nerve synapses may be brought about by: (1) An increase in the flow of nerve impulses through a series of neurons (such as would result from stimulation of the vestibular end organ) or by: (2) Inactivating the enzyme cholinesterase by the administration of such drugs as diisopropyfluorophosphate (D.F.P.), or morphine sulfate. Such an increase in acetylcholine results in an increased rate of flow of nerve stimuli through the series of neurons involved.[9] Morphine is a potent anticholinesterase agent in brain tissue;[10] thereby it produces an increase in the activity of acetylcholine. It is probably, at least in part, by this pharmacodynamic action that morphine stimulates the parasympathetic nervous system as well as the emetic center. This action also permits an increase in the flow of vestibular impulses resulting from the turning tests to reach the emetic center from the vestibular nuclei,[*] with a resulting high incidence of vomiting with vestibular stimulation after morphine. By reducing excessive activity of acetylcholine, dramamine may prevent the "adversive" syndrome by decreasing the increase flow of vestibular impulses on the affected side to a physiological level, thus restoring or maintaining a balance between the two vestibular mechanisms. These considerations indicate that dramamine exerts its effect upon the central part of the vestibular mechanism. We suggest that pharmacologically it may be an antiacetylcholine agent,[†] reducing the effects of excessive acetylcholine activity when such activity exceeds the normal physiological level.

SUMMARY

The effect of dramamine on the vestibular responses to turning was studied in eleven normal subjects. Each subject was turned in a Barany chair following the oral administration of a placebo, and again one week later following the oral administration of 200 mg of dramamine. Audiograms were made after each examination.

There was no statistically significant change in the vestibular responses to turning after dramamine.

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[*] Bard et al[11] have shown that the vestibular cerebellar nuclei are connected with the bulbar emetic center.

Spiegel and Sommer[12] state that the triangular vestibular nucleus is connected by pathways with the vegetative centers in the substantia reticularis.

Borison and Wang[13] have localized the emetic center in the dorsal lateral reticular formation of the medulla of the cat.

[†] In another experiment, we studied the action of dramamine on the inhibiting effect of D.F.P. upon the blood cholinesterase. In two patients 10 cc. of blood were taken for blood plasma and erythrocyte cholinesterase determinations. Immediately after the removal of the blood specimens 100 mg of dramamine were administered orally. One hour later another 100 mg of dramamine were similarly administered. One hour after this, i.e. two hours after the administration of the first 100 mg of dramamine, another 10 cc of blood were taken for cholinesterase determinations. Immediately after the removal of the second 10 cc of blood, 2 mg of D.E.P. in peanut oil were given intramuscularly to each patient. Forty-five minutes after the D.P.F. a third sample of blood was taken for plasma and erythrocyte cholinesterase determination. It was found that dramamine had no effect upon plasma or erythrocyte cholinesterase. After D.F.P. there was a marked reduction in the blood plasma cholinesterase. From this experiment it can be seen that dramamine had no effect upon blood cholinesterase and that it also did not prevent an anticholinesterase agent such as D.F.P. from reducing the blood cholinesterase.

All of the subjects had normal hearing, but following the administration of dramamine there was an appreciable decrease in hearing acuity in two of the subjects. Repetition of the audiograms several days later, in these two subjects, revealed normal hearing. At a later date, the audiograms were repeated on one of these two subjects before and again after the administration of 200 mg of dramamine. Both audiograms were normal. We can offer no explanation for the apparent hearing impairment after the first trial with dramamine. Studies in a larger group of subjects on the effect of dramamine on the cochlear function are necessary in order to establish a definite conclusion in this respect.

The vestibular responses after turning were noted in six of these subjects following the administration of 15 mg of morphine sulfate subcutaneously with 200 mg of dramamine orally. There was no significant change in the vestibular responses following the morphine-dramamine combination. Nausea or vomiting was not present in any of the subjects after the turning tests without and with the morphine-dramamine combination, but following the administration of morphine alone vestibular stimulation produced vomiting in four of the six subjects (66.7%).

Morphine is a stimulant to the parasympathetic nervous system, probably by virtue of the fact that it is a potent brain cholinesterase inhibitor. Dramamine counteracts the emetic effect of morphine perhaps by virtue of the fact that it may be an antiacetylcholine agent.

CONCLUSIONS

1. Dramamine had not effect upon the vestibular responses to turning in normal human subjects. We suggest that dramamine may be an antiacetylcholine agent and thus prevents the emetic effect of morphine which is a potent brain cholinesterase inhibitor.

2. Dramamine apparently exerts an effect in the central nervous system, and it appears to do so wherever there is an increase in acetylcholine activity.

3. Dramamine exerted no effect on blood cholinesterase, nor did it prevent the reduction of blood cholinesterase by diisopropylfluorophosphate (D.F.P.).

4. Dramamine caused a temporary loss of hearing in 18% of our subjects.

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TABLE I—RESPONSES TO TURNING.

SUBJECT	AFTER PLACEBO				AFTER DRAMAMINE, 200 MG.			
	TO RIGHT		TO LEFT		TO RIGHT		TO LEFT	
	NYSTAGMUS Duration in	VERTIGO seconds	NYSTAGMUS Duration in	VERTIGO seconds	NYSTAGMUS Duration in	VERTIGO seconds	NYSTAGMUS Duration in	VERTIGO seconds
1. W. J. E.	26.5	26.5	25	28	26.5	26.5	24.5	24.5
2. F. C.	38	34	35	35	32	30	37	34
3. S. W. C.	25	37.5	25	20	23.5	30	24	34
4. P. E. D.	20	22	19	24	21	24	18	22.5
5. S. E.	24.5	24.5	25.5	25.5	22	22	23.5	23.5
6. R. D. E.	23.5	23.5	26	16.5	25	27	28	24
7. A. K.	28	24	23	19	25	26	25	24
8. S. K.	19	17	28	20	21	18	27	23
9. T. F. H.	15	15	16	16	18	15	15	18
10. C. B. H.	22	27.5	23.5	30	24	28	26	27.75
11. G. W. H.	19	17	22	20	14.5	11	29	28

TABLE II.—RESPONSES TO TURNING.

SUBJECT	AFTER M. S., 15 MG PLUS DRAMAMINE, 200 MG				AFTER M. S., 15 MG PLUS PLACEBO				
	TO RIGHT		TO LEFT		TO RIGHT		TO LEFT		
	NYSTAGMUS Duration in seconds	VERTIGO Duration in seconds	NYSTARMUS Duration in seconds	VERTIGO Duration in seconds	NYSTAGMUS Duration in seconds	VERTIGO Duration in seconds	NYSTAGMUS Duration in seconds	VERTIGO Duration in seconds	
1. W. J. E.	28	28	28.5	29	23	25	could not be determined because of nausea & v.		Vomited
2. F. C.	40	37	35	35	30	30	37	31	Vomited
3. S. W. C.	33	34	22.5	38	25	35	25	39	
4. P. E. D.	20	20	22	22	20.5	22.5	22	22.5	Vomited
5. S. E.	21	21	23	22	20	20	20	20	Vomited
6. R. D. E.	27	27	20	19	25	26	23	25	Nausea

LIX

MALIGNANT MELANOMA OF THE EXTERNAL EAR

REPORT OF 36 CASES TREATED BETWEEN 1928 AND 1944

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AND

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The accumulated experience of the capricious behavior of malignant melanoma as evidenced by previous reports, calls attention to the hazards of delayed and non-radical surgical interventions, and this forms the basis of current trends to apply earlier and more radical surgery in the management of malignant melanoma of the skin. This and related aspects were recently discussed in an analysis of 341 verified cases treated at Radiumhemmet.[19] The present paper embodies the experience gained from treatment of patients with melanoma of the external ear, including the auditory canal and periauricular skin. The report is limited to clinical considerations, and the reader is therefore referred to previous papers regarding other aspects of melanoma.[1, 2, 8, 10-15, 19]

Malignant tumors fairly seldom originate in the skin of the external ear or the acoustic meatus and are consequently rarely treated by otologists, surgeons and radiologists. These tumors are only cursorily mentioned in modern textbooks and by recent authors.[5, 16, 18] It seems, however, to be commonly accepted that malignant tumors located in or near the auditory canal, in the tragal region, and on the posterior surface of the external ear generally present a poorer prognosis than tumors located in other parts of the auricula (Mohs, 1947). The difficulties in the management of carcinomas of the external auditory meatus are well known,[4, 7] and most surgeons seem to hesitate in the choice of radical primary operations. Regarding malignant melanomas of the external ear only a few cases have previously been published,[3, 5] which warrants the present report.

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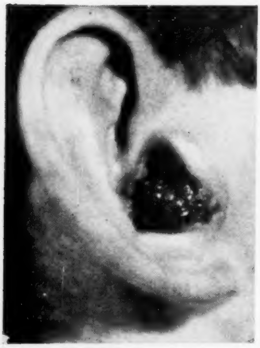
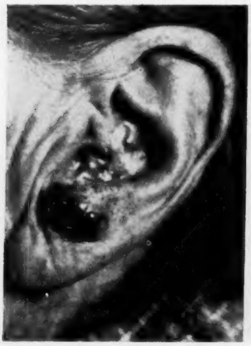


Fig. 1.—Purplish melanoma in a man, aged 25; was in 1936 considered inoperable due to involvement of external auditory canal and cervical nodes. The primary diminished somewhat in size following palliative Roentgen treatment (17 times r = 4,250 r), but the gland deposits remained unaffected. Patient succumbed 8 months after first admission, 32 months after first symptom.



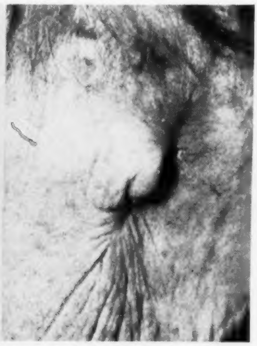


Fig. 2*a*.—Blue-brown melanoma infiltrating part of the auditory canal in a woman, aged 83. Classified as Stage I at admission, October, 1944.

Fig. 2*b*.—Patient's condition after diathermy excision of the auricula and lateral half of auditory canal. No local recurrences. 18 months later patient got node metastases in spite of previous "prophylactic" teleradium treatment (2 fields, 4,500 r each), and finally succumbed 24 months after admission.

MATERIAL AND TREATMENT PRINCIPLES

Our series comprises 36 cases of microscopically verified malignant melanoma treated between 1928 and 1944 at Radiumhemmet in co-operation with other Swedish clinics and hospitals. A prepuberal group including 2 patients (10 and 12 years old, respectively) was distinguished from melanomas in adults. The latter group of 21 males and 13 females presented an average age on admission of 55 years; the youngest adult was 21, the oldest 83 years.

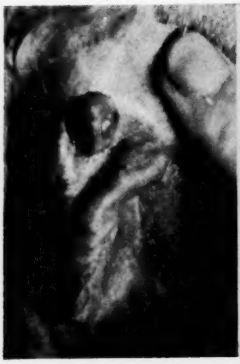
The primary tumors were in general subjected to immediate surgery; the smaller underwent clean excisions, and the larger ones preferably diathermy excisions. All operations were carried out according to conservative surgical principles, and larger mutilating operations were avoided until absolutely indicated. No prophylactic surgical interventions have so far been practiced. Similar restrictive methods were also applied to established lymph node involvement; radical neck dissection was only performed in a few cases during these years. Radiotherapy was mainly used for so-called "prophylatic gland treatment" according to Forssell (1928) in Stage I cases without clinical nodes, or else "prophylactic" teleradium treatment was given (Fig. 2). In Stage II cases, Roentgen or teleradium treatment was further administered to node metastases either pre- or postoperatively, and in advanced cases radiation was used for palliative purposes (Fig. 7 and 8). The actual dosage and methods were described elsewhere.[19] Irradiation treatment as sole therapy was only directed to one single case as illustrated in Fig. 3.

CLINICAL DATA

Classification.—All cases were classified with reference to the extent of the tumor process on admission as ascertained by clinical means. It should be emphasized, however, that such rough clinical screening is naturally impaired by considerable errors. For example, nearby skin deposits are easily overlooked; the true extent of tumor growth in the auditory canal is difficult or impossible to judge previous to operation, and lymph node screening in the retromandibular fossa is further subject to large error on purely anatomical grounds. In spite of this, clinical classification is of prime importance for proper evaluation of methods and results.[19]

Stage I. Localized melanoma confined to the skin. Local recurrences and nearby deposits in cutaneous lymphatics are included.

Stage II. Cases with palpable regional lymph node metastases confined to one gland group only.



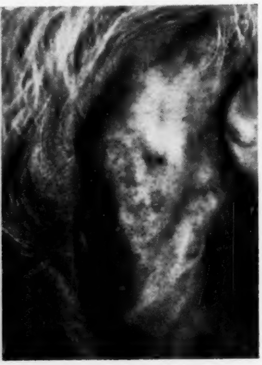
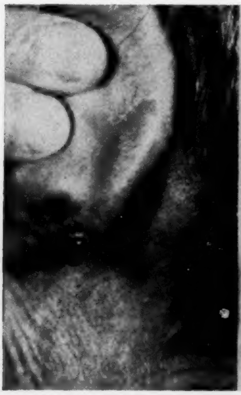


Fig. 3*a*.—Pedunculated non-pigmented melanoma in a man, aged 62. Tumor was implanted with radium needles (40 mg. for 4 hours).

Fig. 3*b*.—Following radium treatment, the tumor area healed without subsequent recurrences in loco. Three years later the same man got a secondary non-pigmented deposit in the preauricular region, which was treated in the same manner on a presumptive diagnosis of squamous-cell carcinoma. Patient dead, probably with cerebral metastases.



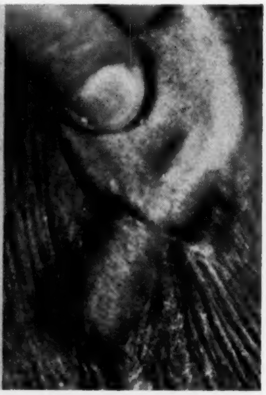


Fig. 4*a*.—Blue-black melanoma in a pre-existing acquired brown naevus. Man, aged 79.

Fig. 4*b*.—Local excision was performed. Patient dead 7 months later without recurrence.

Stage III. Metastatic involvement of two or more groups of glands, and cases with distant metastases evidencing a generalized tumor process.

Distribution.—The localization in this series is shown in Table II. Thus, 19 cases were situated on the external ear (Fig. 2-9), 10 in the adjacent pre- and infra-auricular skin (Fig. 10), and 5 in the skin covering the mastoid process.

Pathology.—Only microscopically verified cases are included. Naevi of questionable malignancy or doubtful histology were excluded. This series contains an unusually large percentage of melanomas poor in pigment red, pink, or purplish in color (18 cases). The rest were brown (9 cases) or black (7 cases).

Precursory lesions.—According to patients' statements, congenital moles were supposed to precede the tumor in only one-third of our cases (11 cases). In the remaining cases the melanoma either developed in acquired naevi (Fig. 4) or in precancerous melanosis.

Dissemination.—Early hematogenous spread from the primary focus without previous or simultaneous lymph node involvement was registered in 3 of our 21 Stage I cases. The corresponding figure in a larger series was 11.5 per cent.[10] In general, lymph node metastases developed prior to generalization. Melanoma localized to the auditory canal showed a pronounced tendency to spread into the tympanic cavity, and those of the mastoid region demonstrated early vascular and lymphatic permeation, which will be further mentioned below. Four cases showed an unusually benign course; two were large and exophytic tumors devoid of metastases (Fig. 6), and two other cases were characterized by long-standing local dissemination in the superficial lymphatic plexus (Fig. 5).

Differential diagnosis.—All pigmented melanomas were correctly diagnosed at the patient's first visit. On the other hand, most non-pigmented ones were misinterpreted until biopsy showed otherwise. The prepuberal melanomas were suspected to be respectively a common wart and a telangiectatic granuloma. Other non-pigmented cases were provisionally diagnosed as atheroma, angioma, fibroma, basal-cell or squamous-cell carcinoma. The correct diagnosis was suspected before biopsy in 5 non-pigmented cases with a history of an earlier pigmented lesion.

Growth stimuli.—As previously reported[2,19] pregnancy is considered to exert a rapid growth-accelerating influence on malignant melanomas. This was found to be the case in one pregnant woman, aged 21 (V.E.E.P., case record No. 15131/1942), in whom large

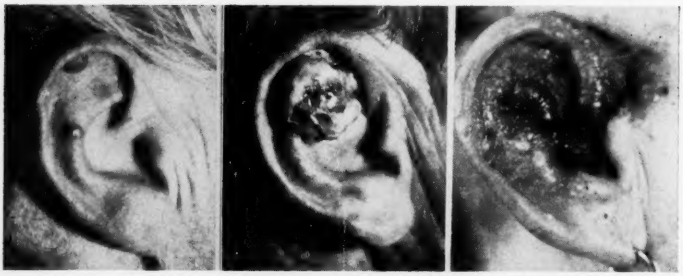


Fig. 5.—The primary melanoma situated on the lobulus was previously excised. One year later, patient got two new foci of melanoma growing in the cutaneous lymphatics. Died free of symptoms four years after last excision. These recurrences, which could easily have produced hematogenous spread, could have been avoided by extirpation of the ear at the first instance.

Fig. 6.—Large exophytic non-pigmented melanoma in a man, aged 49. Following local excision this patient has been free from symptoms for 9 years.

Fig. 7.—Extensive rapidly growing and metastasizing non-pigmented melanoma in a woman, aged 71, First sign of tumor 2 months before admission. Considered inoperable due to involvement of auditory canal. Palliative teleradium treatment without apparent effect. Died 4 months after admission.

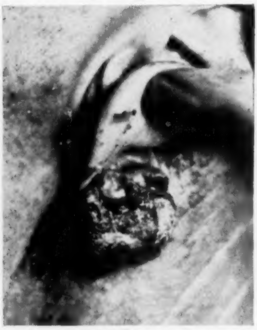


Fig. 8.—A blue-black infra-auricular melanoma in a man, aged 61, running a rapidly fatal course. First sign of tumor 6 months previously, when he noticed a change in a pre-existing congenital black mole.

gland metastases and generalization developed within a few months of the first sign of melanoma.

A detailed account of the material and results with reference to different groups, localizations, and stages of disease on admission is presented in Tables I and II, and the following statements seem justified.

TABLE I
CLINICAL STAGES ON ADMISSION AND 5-YEAR SURVIVAL RATE

GROUP	STAGE	NO. OF CASES	LIVING FREE OF SYMPTOMS	PERCENTAGE 5-YEAR SURVIVAL RATE
PREPUBERAL	Stage I	2	2	100
ADULT	Stage I	21	11	52
	Stage II	8	0	0
	Stage III	5	0	0

The relative innocence of melanomas with microscopic characteristics indicating malignancy appearing in prepuberal children warrants their separate classification, which was known previously.[11, 13, 14, 19]

In our series of adult patients 21 cases out of 34 (62 per cent) looked for medical help as early as in Stage I, and further in the majority of these patients (16 cases) the tumor had a rather favorable location (Table II). A remarkably high five-year survival rate was found: the overall five-year survival rate (uncorrected for intercurrent deaths) was 32 per cent in the adult group of patients, and 52 per cent in Stage I patients only. It should further be noticed, that cures were only obtained in patients admitted in Stage I; no single cure was obtained in Stage II patients, which already on admission had demonstrable lymph node metastases. The patient's outlook with reference to the actual location of the melanoma is much poorer when the tumor is situated in the central part of the auricula and in the skin covering the mastoid region (Table II).

A comparative evaluation of the methods hitherto used in treatment of the primary Stage I melanomas is shown in Table III. Small circumscribed clean excisions (Fig. 4), followed by suture, resulted in a larger number of subsequent local recurrences in the scar (Fig. 9, and the case report below), than the larger block excisions by means of diathermy. In the choice between these two methods, the latter is therefore to be preferred.

The following case history illustrates the hazards in limited non-radical interventions, and also the difficulties in the management of melanoma involving the central part of the auricula:

TABLE II
RESULTS IN AURICULAR MELANOMAS TREATED AT RADIUMHEMMET FROM 1928 TO 1944

GROUP AND SITE OF PRIMARY TUMOR		TOTAL NO. OF CASES	DIED OF MELANOMA	INTERCURRENT DEATHS WITHIN 5 YEARS (INDETERMINATE CASES)	LIVING FREE OF SYMPTOMS 5 YEARS OR MORE	PERCENTAGE 5-YEAR SURVIVAL RATE AND END-RESULTS AT 1949
PREPUBERAL GROUP						
External ear		2			2	100
ADULT GROUP						
Central part of auricula, including the external auditory canal, concha, tragus, and antitragus	Stage I	2	2	...	...	0
	Stage II	2	2	...	...	0
Periphery of auricula exclusive of central part	Stage I	11	2	2	7	63
	Stage II	2	1	1	...	0
	Stage III	2	2	...	...	0
Pre- and infra-auricular region	Stage I	5	1	1	3	60
	Stage II	3	3	...	...	0
	Stage III	2	2	...	...	0
Retroauricular region	Stage I	3	2	...	1	33
	Stage II	1	1	...	...	0
	Stage III	1	1	...	...	0
All adult cases		34	19	4	11	32

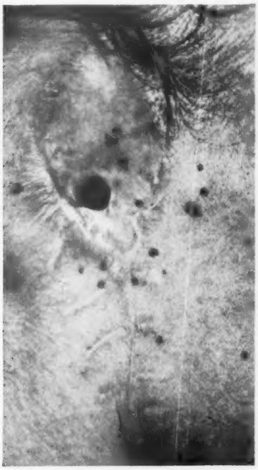


Fig. 9.—Patient's condition February, 1940; see case report in text.

CASE REPORT. A. M. K. A woman, aged 30, noticed a brown spot on her right lobulus in 1937. This was treated by electrodesiccation at a Stockholm Hospital. The scar later became reddish, covered by a crust, and for some time she had "eczema." On admission to Radiumhemmet in March, 1939, the biopsy report was "Naevus of suspected malignancy." No secondary lesions or lymph nodes were observed. A local excision was done in May, 1939; the report was "malignant melanoma." In July, 1939, she reappeared with a recurrence in the scar, and two additional nonpigmented melanomas in the antitragal region and near the helix. The external ear was then removed, but the external auditory meatus was left. The pathologists found additional melanoma foci in subcutaneous vessels. In September, 1939 there were several recurrences in the skin of the external meatus, and in addition two enlaryged lymph nodes. At subsequent reoperation, large areas of periauricular skin were removed, the skin lining the meatus was removed by curettage, and finally a radical neck dissection was done. Microscopy revealed small cutaneous foci of tumor, but no glandular deposits were found. In January, 1940, new foci appeared in the periauricular previously grafted skin (Fig. 9), and later as well in the auditory meatus. In March, 1940, she received massive teleradium and radium treatment, but no radical interventions were planned. Finally, in November, 1940, radical mastoidectomy revealed large tumor vegetations in the innermost part of the external meatus, and further throughout

TABLE III
TREATMENT METHODS AND RESULTS IN STAGE I PATIENTS

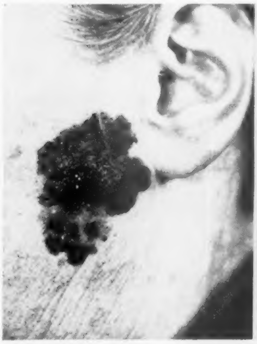
TREATMENT	NO. OF CASES	NO. OF SATISFACTORY LOCAL RESULTS	NO. OF CASES WITH SUBSEQUENT LOCAL RECURRENCE	PERCENTAGE OF LOCAL RECURRENCE	NO. OF 5-YEAR CURES
CLEAN EXCISIONS					
Small excisions	9[a]	5	4	45%	7 out of 10
Large excisions + plastic repair	1[b]	1	0	—	
DIATHERMY EXCISIONS (more advanced cases)					
Without subsequent local irradiation	9[c]	7	2	22%	4 out of 10
With subsequent irradiation	1[d]	1	0	—	
IRRADIATION TREATMENT ONLY					
Implantation of radium needles (Fig. 3)	1	0	1	—	0

[a] 5-year survival rate 6 out of 9 patients

[b] 5-year cure obtained

[c] 5-year cure only obtained in 3 patients (see text)

[d] 5-year cure obtained



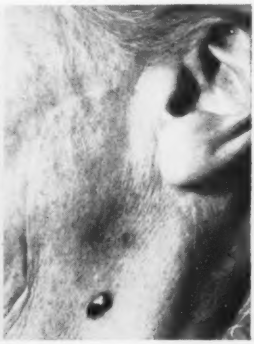


Fig. 10*a*.—This woman, aged 69, had for 15 years a slowly growing "precancerous melanosis", in which a reddish elevated melanoma developed.

Fig. 10*b*.—Following wide surgical excision and plastic repair patient has now been well since 8 years.

the tympanic cavity. A radical intervention was attempted; signs of generalization developed and the patient died with metastases in February, 1941.

With a view to a greater salvage in future of our Stage I patients, a short compilation of our failures might convey some useful information. According to the figures in Table III, seven patients of our Stage I series have actually succumbed with melanoma following our local excisions described above. The subsequent course of these cases and the supposed errors made in treatment will be stated below:

Supposed haematogenous spread from primary tumor or unknown foci:
following local diathermy excisions .... 2 cases
following radium implantation (Fig. 3) .... 1 case

Delayed or incomplete operation of primary:
involving the central part of auricula .... 2 cases

Gland metastases treated by teleradium instead of radical dissection: .... 2 cases

This seems to indicate, that in the first two instances compiled above, no doctor would have been able to bring help; but in the remaining five cases the patients' outlook might have been better if more radical interventions had been planned at an earlier date.

TABLE IV

SURVIVAL RATES IN RELATION TO SIZE OF PRIMARY IN ALL STAGE I MELANOMAS

SIZE OF PRIMARY MELANOMA	TOTAL NO. OF CASES	FAILURES (DEAD WITH MELANOMA)		INTERCURRENT DEATHS IN PATIENTS FREE FROM TUMOR	LIVING FREE OF SYMPTOMS	
		NO. OF CASES	PERCENTAGE		NO. OF CASES	PERCENTAGE
Less than 10 mm	8	2[a]	(25)	2	4	(50)
10 - 20 mm	9	3[b]	(33)	1	5	(55)
More than 20 mm	4	2[c]	(50)	--	2	(50)

[a] Early haematogenous spread from primary focus in 1 case

[b] ” ” ” ” ” ” ” 1 case

[c] ” ” ” ” ” ” ” 1 case

For the purpose of the records, the clinical course of our Stage I melanomas was further correlated with the approximate size of the primaries. In Table IV the previously well known heterogeneity in biological capacities and malignancy exhibited by different melanomas in man is again evidenced. Actually, several types may be distinguished, and among them we meet a few large exophytic tumors with a comparatively slow tendency to infiltration and dissemination. This type is illustrated in Fig. 6. The practical consequence is that a reliable prognosis can in general not be based on the size of the primary melanoma.

The present figures are too small to permit conclusions regarding the evaluation of methods used for treatment of lymph node involvement. In Table V, both Stage I and Stage II patients with glandular metastases are included. The figures, however, show that present results are very poor. Only two Stage I patients presenting early involvement of one single node have been salvaged by gland dissection. The case records thus indicate that the lymph node barrier is penetrated much more rapidly than was believed,[19] which in turn necessitates earlier surgical interventions than hitherto practiced.

COMMENTS AND RECOMMENDATIONS

Circumscribed excisions of primary melanomas evidently give rise to a fairly large number of local recurrences and secondary growths in the adjacent skin, calling for extensive reoperations and involving loss of time and decrease in survival rate. This may in the future be avoided by adopting wider excisions preferably by means of electrosurgery.

Our expectant attitude toward late lymph node involvement in patients admitted as Stage I implies that pertinent metastases have to reach a palpable magnitude before radical interventions are planned. The evident deficiency (Table V) in such treatment necessitates more active and radical surgical interventions at an earlier date, if that will be possible in the future. It should further be realized that the anatomical conditions in this region as regards the excessive vascularization of the external meatus, as well as the concealed location of the superior deep cervical glands, requires special attention in planning effective operative methods.

The deduced recommendations suggested below are in principle in agreement with those given by several previous authors on melanoma.[1, 11, 15] Due regard is moreover paid to the anatomical conditions of the auricular region. According to our opinion, the external ear can in most persons be sacrificed without too great dis-

TABLE V
SURVEY OF TREATMENT METHODS AND RESULTS IN STAGE I AND II PATIENTS WITH LYMPH NODE INVOLVEMENT

STAGE AND TREATMENT	NO. OF CASES	DIED OF MELANOMA	LIVING FREE OF SYMPTOMS 5 YEARS	COMMENTS
Stage I Patients				
Prophylactic gland dissection+post-operative tele-radium	1	1	0	Haematogenous spread
Gland dissection only	2		2	One single node involved
Preoperative tele-radium + gland dissection	1	1	0	Operation delayed
Palliative irradiation	2	2	0	Considered inoperable
	6	4	2	
Stage II Patients				
Preoperative tele-radium + gland dissection	2	2	0	
Gland dissection + postoperative teleradium	2	2	0	One case had advanced node involvement
Palliative irradiation	2	2	0	Inoperable
	6	6	0	

comfort. Present progress in prosthesis can provide many possibilities for masking the defect. In this region the radical dissection in continuity of both primary tumor and eventual lymph node involvement is further facilitated by the short distance between the external ear and the cervical nodes. The following recommendations are likewise applicable to other high-grade malignancies affecting the central part of the auricula or the acoustic meatus.

We feel that the severity of the disease under question justifies our recommendation to inform the patients about the possible risks inherent in limited non-radical surgical interventions.

PREPUBERAL MELANOMAS

The relative innocence justifies local radical excision and careful follow-up.

MELANOMAS IN ADULTS

A. Primary melanoma located in central part of auricula and large melanomas located in any part of auricula:

1. *Biopsy*: This should be done in the operating room by the surgeon preferably by diathermy, and subjected to immediate microscopy.

2. *Radical Extirpation*: Once the diagnosis is verified, the external ear together with the cartilaginous part of the external meatus and the tragal region, as well as the surrounding periauricular skin, adipose tissue and deep fasciae, should be removed *en bloque*. If melanoma is found or suspected in the lining of the external meatus a radical mastoidectomy will be necessary in order to secure removal of the meatus *en bloque*.

3. *Gland Dissection*: In this group radical neck dissection should be performed in cases both with and without (so-called "prophylactic" dissection) palpable nodes. Particular care should be taken to remove the uppermost deep cervical glands surrounding the carotid artery.

B. Small melanomas located in peripheral parts of the auricula:

1. *Excision*: A large radical electro-excision should be made.

2. *Microscopy*: Special care should be taken to prepare sections from different parts of the specimen in order to find microscopic deposits in adjacent lymphatics.

3. *Follow-up*: Careful follow-up of patient.

4. *Recurrences*: Near-by skin deposits indicate spread and under such conditions radical operation plus subsequent neck dissection *en bloque* should be performed according to item A (above).

C. Small melanomas located in pre-, infra-, or post-auricular skins regions:

1. *Excision*: Radical excision with removal of suitable skin area together with the underlying adipose tissue and deep fasciae *en bloque*.

2.-4. Same recommendations as item B (above).

The question whether the steps mentioned under item A should be performed at one operation or, if the neck should be dissected at a subsequent operation some weeks later was discussed by Pack, who emphasized a number of factors influencing this matter. With reference to the auricular melanomas we consider long delay before gland dissection is performed to be hazardous. Patients volunteering ability to return for frequent follow-up examinations should be informed of the risks associated with such delay and of the inadequacy of clinical lymph node screening by palpation in this region.

The scheme outlined above is expected to meet the requirements for earlier and more radical surgical treatment of auricular melanomas than previous conservative and expectant methods could provide. Particularly in Group A cases a larger survival rate can be expected, but we are of course at present unable to predict the possible increase in salvage. It has however to be recalled that similar principles in the hands of Pack and others have been reported to give considerable increase in the cure rate of melanomas in other locations.[1, 13, 15]

SUMMARY

During the years 1928 through 1944 thirty-six verified cases of malignant melanoma originating in the auricular skin region, including the external auditory meatus and the periauricular skin, have been treated at the Radiumhemmet. A brief review is presented on pertinent clinical data, treatment methods hitherto used, and the results obtained. Conservative surgical methods, characterized by limited excisions of primary melanoma and combined lymph node treatment (radiotherapy and surgery), have resulted in a 52 per cent five-year survival rate in Stage I patients. The over-all five year survival rate was 32 per cent. Previous principles of treat-

ment are shortly evaluated and the suggestions are advanced (1) that by adopting more radical and earlier surgical interventions a number of local recurrences and cutaneous satellites would be avoided; and (2) that a number of lymph node metastases appearing in patients admitted as Stage I cases would possibly be avoided by applying "prophylactic" neck dissections. Recommendations based on the present experience are advanced, and an increase in survival rate is consequently expected.

Sahlgrenska Sjukhuset.

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LX

THE TONSIL AFTER RADIUM IRRADIATION

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Numerous papers have appeared advocating the use of irradiation of tonsils in lieu of surgical removal. However, the current treatment is tonsillectomy, excluding such cases as hemophilia, certain heart, lung and kidney diseases, and those in which the patient refused operation. Nuzum[1] found that irradiation was effective in reducing the size of tonsils in only 16 per cent of his subjects, and that hemolytic streptococci, the predominant organisms in chronically infected tonsils, were permanently removed in only 50 per cent of 32 patients treated with radium. It is noteworthy also that Bloom in his recent book on the histopathology of irradiation[2] does not mention its effects on tonsils.

It is thought worthwhile to report the histopathological findings of a case in which surgical removal of the tonsils was done twenty years after radium irradiation. These findings support the contention that surgical excision is more satisfactory than irradiation.

A 52 year old white female sought our advice for repeated attacks of sore throat followed by pains in the joints. She stated that in 1928 she received radium irradiation of her tonsils. One radon seed had been inserted into each tonsil for three days. Since each seed gave off 55,6 mc hours daily, the patient had received 166,8 mc hours to each tonsil.

When seen by us her physical examination was negative except for large and chronically inflamed tonsils. These showed numerous crypts filled with cheesy material. The anterior pillars were injected and the lingual lymphoid tissue was grossly inflamed.

A tonsillectomy was done and numerous strong adhesions were encountered. The postoperative course was free of complications and recovery was uneventful.

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The tonsils were well encapsulated structures of mature lymphoid tissue showing the usual architecture. The stratified squamous epithelium of the free surface was mildly hyperkeratotic. The lymphatic elements showed a moderate amount of hyperplasia with an increase in the number of germinal centers. Many mononuclear phagocytes were scattered throughout the sinusoids. Fibroplasia with a dense proliferation of scar tissue was a prominent feature and this had replaced a great deal of the parenchymatous tissue. The crypts were dilated, but found to be free of foreign material. Occasional islands of metaplastic hyaline cartilage were encountered within the fibrous tissue. The vessel walls appeared generally thicker than usual with narrowing of their lumina. It was impossible to discern however, whether this was the result of atrophy or irradiation. The diagnosis was atrophy of tonsils with probable radiation sclerosis.

Summary: The histopathological findings of previously irradiated tonsils are reported.

STATION HOSPITAL U. S. MILITARY ACADEMY.

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LXI

ANGIO-FIBROSARCOMA OF THE ETHMOID AND FRONTAL SINUSES COMPLICATED BY OSTEOMYELITIS OF THE FRONTAL BONE AND EPIDURAL ABSCESS

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In a review of the literature on angiosarcoma, Kinkade[1] stresses the fact that the disease is not so rare as is generally believed, provided cases described by various names but of similar histologic picture are included in the statistical study. He thus includes in his review hemangiosarcoma, hemangioendothelioma, angioepithelioma, and angioblastic sarcoma. He suggests the more comprehensive name "mesenchynoma of prevalently vascular appearance." His review of cases extends over a period of fifteen years, from 1934 to 1949. It is noted that in this series there is not a single case involving the sinuses. The cases recorded were of the central nervous system, spleen, liver, mandible, mouth, thyroid gland, retroperitoneal space, postnasal space, lip, lung, bone, mediastinum, intestine, lymphoid tissue, eyelid, kidney, and muscle.

Freilich and Coe[2] reviewed the literature on angiosarcoma from 1918 to 1936. During this period 29 cases were reported, only one being a case of angiosarcoma of the sinuses. This case was reported by Pagano,[3] and occurred in the maxillary sinus in a 32 year old female with symptoms of swelling, pain, and nasal discharge. Surgery was performed with recovery. The remaining cases were of the pancreas, jejunum, femur, kidney, retroperitoneal space, spleen, liver, clavicle, mediastinum, muscle, forehead, lip, tongue, perineum, ribs, brain, lung, and thymus.

From the division of Otolaryngology, Grasslands Hospital, Valhalla, N. Y.

The case of Freilich and Coe[2] was of the scapula and occurred in a 60 year old man with metastasis to the spleen, liver and axillary, supraclavicular and retroperitoneal nodes. The pathology is given as "great cellularity, numerous blood vessels and a distinct tendency of the undifferentiated mesenchyme to vasoformation."

Stout[4] describes the tumor as being made up of congeries of vascular tubes which have a marked tendency to anastomose and are lined with hyperchromatic, atypical endothelial cells. These may be of any shape or size. They may form a single layer, be heaped up in several layers, or even multiply to such a degree that the vascular tubes are completely obscured when ordinary stains are used and can only be demonstrated by silver connective tissue impregnation.

Simon[5] reports a case of hemangioendothelial sarcoma of the thyroid occurring in a 58 year old man. The tumor invaded the trachea and metastasized to the cervical and medistinal nodes.

CASE REPORT

T. W., colored male, age 27, was admitted to the hospital May 8, 1946, with the chief complaint of headache. The history revealed that eight weeks prior to admission he suddenly developed severe frontal headache, nausea, and vomiting. These symptoms subsided in three or four days only to recur after an interval of a few days. The attacks continued to recur and subside at similar intervals. One month after the onset he suddenly developed chills and fever. The temperature rose to 101°F. A swelling appeared on the right side of his forehead. He stated that a sulfonimide preparation was prescribed by a physician and this he took in dosages of two tablets three times a day for one week. At the end of this time the temperature was normal, the swelling on the forehead disappeared but the headache remained. Three weeks later, that is several days prior to this admission, he again had chills and fever and once again a sulfonimide preparation was prescribed by his doctor, which he had been taking up to the time of admission.

Physical examination on admission: The patient did not appear to be acutely ill. He was somewhat dull mentally. There was a slight swelling over the right frontal region which was tender on palpation. It was localized to the right frontal bone supraorbitally, but had no sharp line of demarcation and tapered off gradually. There was no fluctuation. It was firm. Palpation of the floor of the frontal sinus did not elicit tenderness. The right nasal fossa revealed a large mass occupying the greater part of the nasal cavity. It was polypoid in appearance, rather firm, smooth, not ulcerated, and appearing to originate in the middle meatus. The left nasal

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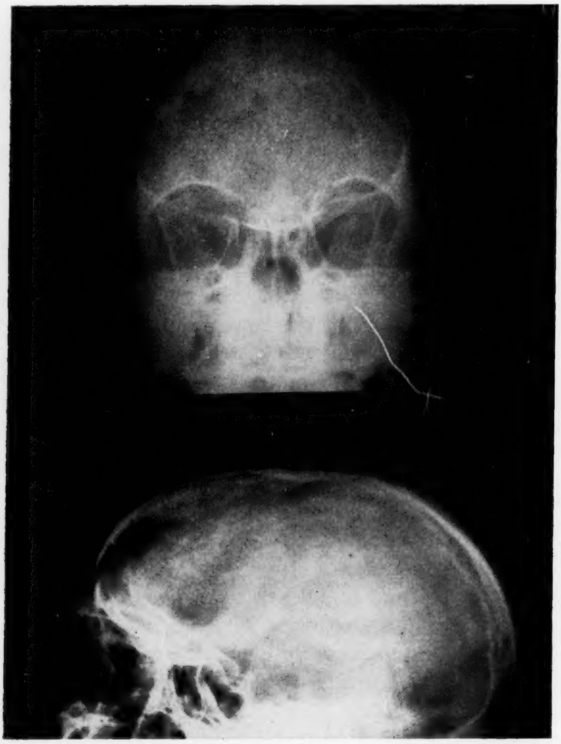

Fig. 1.—X-rays taken prior to operation showing cloudiness of all sinuses and bony rarefaction in frontal sinuses.

fossa was clear. After applying shrinking solution (Adrenalin Hydrochloride 1-1000) to the nasal cavity, mucopurulent discharge was seen in the middle meatus on the right side. Posterior rhinoscopy revealed the same mass extending slightly beyond the choano into the nasopharynx. Mucopurulent discharge was seen in the nasopharynx.

X-ray examination of the nasal accessory sinuses and skull (Fig. 1) showed a fairly marked clouding of the right antrum and less marked clouding of the left antrum. There was a diffuse haze in the region of the right ethmoid cell group and in both frontals. Superior to the upper margin of the right frontal sinus, there was a small area of decreased tissue density, interpreted as representing a rarifying osteitis. X-ray of the chest was negative.

Laboratory data: Blood count revealed 4.65 million red blood cells, 10 gm hemoglobin, 5000 white blood cells, the differential count being 70% segmented forms, 4% monocytes, and 26% lymphocytes. Urinalysis was negative. Blood sugar was 100, NPN 35, chlorides 470, total proteins 6.8%. Blood Wasserman was negative. Blood cultures were negative. The spinal fluid examination showed 22 white blood cells, 9 red blood cells and a trace of globulin, sugar 65, total proteins 24, chlorides 720, and colloidal gold 1 122 110 000. Spinal fluid Wasserman was negative.

Neurological examination showed some hyperesthesia in the right frontal region, slight restriction of associated movements in the right arm, and weakness in the muscles of the right foot and leg with exaggerated right ankle jerk and failure of plantar flexion on the right side. The eye grounds were normal.

A diagnosis of suppurative sinusitis with nasal polyps, complicated by osteomyelitis of the frontal bone and possible brain abscess, was made. The symptoms began to improve shortly after admission. The temperature became normal the day after admission. Headache, nausea, and vomiting were mild and occasional. The patient was scheduled for right nasal polypectomy in order to establish better drainage from the sinuses. Following preparation of the nasal fossa with 10% cocaine—1:1000 adrenalin solution, the mass was grasped with a nasal snare. There was profuse bleeding as soon as it was touched, and it felt rather solid in consistency. Because of this it became apparent that we were dealing with more than a benign polyp, and a biopsy alone was performed. Profuse bleeding occurred but it was easily controlled. Following the procedure there was a decrease in severity of the headaches, and the nausea and vomiting ceased.

On June 6, 1946, an external radical frontal and ethmoid operation was performed. A right supraorbital incision through the skin and periosteum was made.

Following elevation of the periosteum two areas of bony dehiscence were seen in the right orbit. One was in the floor of the frontal sinus and the other in the lamina papyrocea of the ethmoid. A soft mass was seen projecting into the orbit from these sinuses through each dehiscence. On opening the right frontal and ethmoid sinuses this soft vascular tumor was seen to fill each sinus completely, forming one continuous mass, and having eroded the bone in its path. The superficial surface of the anterior plate of the right frontal sinus was intact and appeared normal grossly, but the diploic region and the deep surface were grossly necrotic and purulent. The anterior plate of the frontal sinus was, therefore, removed completely. The mass that filled the frontal sinus was next dealt with. Its posterior surface was found to be adherent to the dura, the posterior bony plate of the right frontal sinus having been almost completely eroded except for a small margin of bone one cm in diameter. The mass was separated from its bed and from the dura by blunt dissection. A considerable quantity of pus was then seen to escape from a pocket above the sinus between the frontal bone and dura (epidural abscess). The intersinus septum was found partly destroyed by the tumor which extended into the left frontal sinus. A small portion of the posterior bony plate of the left frontal sinus was necrotic, but the dura was not adherent to the mass. Next, the right ethmoids were exenterated including the tumor.

There was loss of considerable quantity of blood during the course of the operation and this was replaced with whole blood by transfusion. The osteomyelitic process was too extensive and could not be handled through the type of incision made. Furthermore, the patient was in only fair condition and had already been under anesthesia a considerable length of time. It was, therefore, decided to complete the operation at another stage. Hemostasis was established and the wound was dressed without closure. Three days later, frontal osteotomy was performed through an incision in the scalp beyond the hairline, as it was obvious that the infection of the bone extended beyond the confines of the sinuses. At this operation a large portion of the right frontal bone, small portions of the right parietal and left frontal bones were removed. This included all of the infected bone and a margin about one inch wide of grossly healthy appearing bone. Again, the wound was dressed without closure. Secondary closure was performed nine days later. In another week the wound was completely healed. Except for an elevation of temperature to 100° F. for about five days after operation, the temperature remained normal. Chemotherapy and antibiotics consisted of 1 gm of sulfadiazine every four hours from June 5, 1946, to June 8, 1946, totalling 84 gms, and penicillin intramuscularly, 50,000 units every three hours from June 5, 1946 to June 14, 1946, and 20,000 units every three hours from June 15, 1946 to June 25, 1946, totalling 5.5 million units.

The specimen was submitted to Dr. Andrew A. Eggston, Pathologist, Manhattan Eye, Ear, and Throat Hospital, whose report follows: "Specimen consists of numerous masses of rather firm, greyish white homogenous tissue, measuring 7x6x5 cm. On sectioning, the cut surface was rather firm. The cut surface is greyish white and shows some hemorrhagic areas. Microscopic examination reveals the tissue is composed of numerous spindle and stellate cells with vesicular

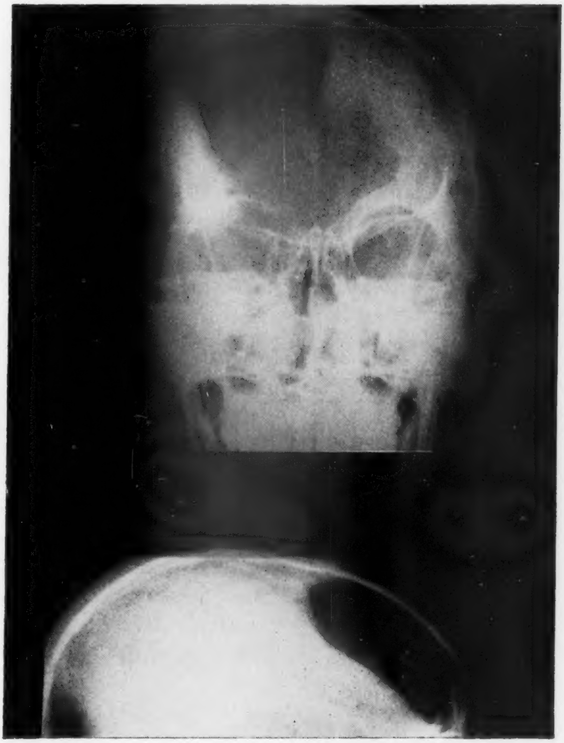

Fig. 2.—X-rays taken postoperatively showing defects in bone (operative). *A.* A-P view. *B.* Lateral view.

nuclei and a large amount of intercellular fibrillae. In some areas the cells are quite well differentiated; in other areas the cells are fibroblastic. There is some mitosis. There are numerous small blood vessels and some cavernous blood spaces are present. Some of the arteries show arteriolar fibrosis. The blood vessels show numerous undifferentiated mesenchymal cells. In other areas the cells are fibrotic and there are some collagen fibres present. Some of the blood vessel walls are close in apposition to the cellular structures and are without definite musculature. Diagnosis: Angiofibrosarcoma."

The postoperative course was uneventful. All wounds healed well. In addition to the usual postoperative care, the right antrum was irrigated several times until the washings were clear. X-rays following lipiodol instillation showed no filling defect in the antrum.

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The patient was discharged July 11, 1946, at which time the nasal fossae appeared clear, and there were no subjective complaints. There was, of course, the bony defect (Fig. 2).

X-ray therapy, which was started the day before discharge, was continued as outpatient treatment and was completed September 4, 1946. It consisted of doses of 150r, using a ¾" copper filter, applied to the right and left frontal and ethmoid sinuses, and given three times weekly for a total of 3600r to each side.

The patient is seen at regular intervals and thus far, four years postoperatively, there is no evidence of recurrence. The bony defect is smaller, some regeneration having taken place.

SUMMARY

A case of angiofibrosarcoma involving the frontal and ethmoid sinuses, complicated by osteomylitis of the frontal bone and epidural abscess is presented. Brief reference is made to the literature. This type of tumor occurring in the sinuses is extremely rare as indicated from the review of the literature. The masking effect of sulfonimides is well illustrated in this case by the subsidence of symptoms notwithstanding the progressive pathologic changes. Treatment consisted of radical surgery, sulfadiazine, penicillin, and x-ray therapy. There is no evidence of recurrence four years postoperatively.

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LXII

FURTHER INVESTIGATIONS ON THE PHYSIOLOGY OF THE LABYRINTHINE FLUIDS

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NEW YORK, N. Y.

INTRODUCTION

In a previous paper, the authors reported preliminary experiments designed to investigate some questions pertaining to the physiology of the labyrinthine fluids (Altmann and Waltner,[2] 1947). Using the method employed by Weed[21] in his studies of the resorption of the cerebrospinal fluid, solutions of iron-ammonium citrate and potassium ferrocyanide in various concentrations and oxalated chicken blood were introduced into the subarachnoid space of rabbits and traced in two instances into the labyrinth.

It was concluded that the iron salts are carried from the subarachnoid space into the cochlear aquaeduct and the fundus of the internal auditory meatus. It was assumed that near the inner opening of the aquaeduct the movement is slowed down, but that the salts may, nevertheless, enter the scala tympani within a few minutes. From the scala tympani the salts seem to be carried upward to the helicotrema and then downward into the scala vestibuli. Only a relatively small amount reaches the vestibule.

Within the cochlea a movement was believed to occur from the scala vestibuli through Reissner's membrane and, possibly, also from the scala tympani through the basilar membrane into the cochlear duct.

The distribution of the Prussian blue granules was taken as an indication that the greater part of the iron salts is resorbed within the cochlea into the tissue spaces of the spiral ligament and the crista spiralis and from there eventually into the venous system. Only a

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The study was assisted by grants from the Research Council of the American Otological Society and the Hayden-Coakley Fund.

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small amount of the solution which has penetrated into the cochlear duct is carried down into the saccule and from there into the endolymphatic sac. This small part was believed to be resorbed through the wall of the latter. Another weaker current seems to carry the salts from the fundus of the internal auditory meatus along the nerve fibers into the subepithelial tissue of the various endorgans.

The authors concluded furthermore, that in rabbits the perilymph is derived mainly from the cerebrospinal fluid which reaches the perilymphatic spaces through the aquaeductus cochleae and, to some extent, the internal auditory meatus. Some of the perilymph is possibly derived from the endolymph and vice versa. It was assumed that if the pathway which is followed by the salts is indicative of the physiological currents within the perilymph, the resorption of the latter in the spiral ligament and in the crista spiralis occurs at a rapid pace.

This paper reports the results of further experiments in rabbits, cats and monkeys, and adds some additional knowledge to our still fragmentary knowledge of the physiology of the labyrinthine fluids.

EXPERIMENTS IN RABBITS

In 15 rabbits, after withdrawal of 0.5-0.75 cc of spinal fluid, 0.2-1.0 cc of a 1 per cent or 5 per cent solution of potassium ferrocyanide and of iron-ammonium citrate was injected into the cerebellomedullar cysterna. In view of the possibility of a rapid resorption of the iron salts in the cochlea, the animals were killed 1-15 minutes after the injection. The temporal bones were fixed in a 10 per cent solution of formalin to which 5 per cent hydrochloric acid was added. Table 1 gives the details.

On examination of the 5 rabbits injected with 1 per cent solution, granules were found in the outer half of the cochlear aquaeducts in all of them, although in very small numbers in one. The fundi of the internal auditory meatus contained larger numbers of granules in three rabbits, and very few granules in two of them.

In only one instance (rabbit 121) within 5 minutes after the injection granules had penetrated through the entire cochlear aquaeduct into the scala tympani of the lowest turn and the lower half of the second cochlear turn. Granules were also found in the spiral ligament, mostly in the lumen of small blood vessels and in lesser numbers between the cells of the organ of Corti and in the tissue spaces of the crista spiralis. The fundus of the internal auditory meatus contained very few granules. In three rabbits (120, 121, 122) identical areas on one side, in the subepithelial tissue of the medial wall of the proximal part of the intradural portion of the endolymphatic sac, were found to contain granules. Granules were also present in the adjacent portion of the dura; there were also more numerous granules in the subarachnoideal space corresponding to this area. No granules were found in the lateral wall of the sac, in the epithelial lining, or in the lumen. The fact that the granules were only found in one circumscribed area of the dura and that their number gradually decreased from the subarachnoid space towards the medial wall of the endolymphatic sac favors the assumption of an intravital movement of the salts in this direction in the animals examined.

In all the ten rabbits injected with the 5 per cent iron-salt solution, granules were found in the cochlear aquaeducts and the fundi of the internal auditory meatus. In seven animals (127, 128, 134, 125, 126, 131, 130) the iron salts had penetrated through the cochlear aquaeduct into the cochlea.

The findings in these seven cases, arranged according to the extent of penetration of the iron salts into the inner ear, were briefly as follows:

Rabbit 127. Prussian blue granules were found in the scala tympani of all turns up to the helicotrema and down the scala vestibuli up to the vestibule. On one side, there were also a few in the perilymphatic space of the vestibule and in the lumen of some small trabecular vessels. They are seen furthermore in the tissue spaces and the lumen of some blood vessels in the spiral ligament, particularly near the prominentia spiralis, in tissue spaces and vessels of the crista spiralis, in the various spaces within the organ of Corti, and extending from there along the perineural spaces of the cochlear nerve fibers within the lamina spiralis ossea into the spiral ganglion. In the scala vestibuli, the granules were seen in larger numbers on the outer (vestibular) surface of Reissner's membrane, on the upper surface of the crista spiralis and on the surface of the uppermost parts of the spiral ligament above the point of insertion of Reissner's membrane. Granules were seen also in the lumina and the perivascular spaces of some of the vessels of the interscalar septa and of the modiolus.

There was an accumulation of granules in the fundus of the internal auditory meatus extending along the perineural spaces of the cochlear nerve into the spiral ganglion of the lower and, to a lesser extent, of the upper basal turn. Others could be traced along the perineural spaces of the saccular nerve into the macula sacculi of one side up to the epithelium of the latter, but not into the otolithic membrane or the lumen. Other granules extended into the stroma of one crista ampullaris and could also be seen in the lumen of some small vessels in this area. There were none in the endolymphatic duct or sac.

Rabbit 130. Prussian blue granules were found on one side in the scala tympani up to the helicotrema, and a few of them in the lumen of small vessels of the perilymphatic tissue of one semicircular canal. Within the scala tympani and the cochlear duct, there was a similar distribution as in the preceding case; they were, however, more numerous within and around the interscalar and modiolar vessels, and some of them were seen also within the lumen of some vessels in the fundus of the internal auditory meatus.

On the other side, the granules, much less numerous in the aquaeductus cochleae, extended from there into the scala tympani of the first and second turn. A few could be seen within the spiral ligament, the organ of Corti, the prominentia, and crista spiralis.

Rabbit 126. Granules extended through the scala tympani of all the turns into the scala vestibuli of the uppermost turn. Otherwise, the distribution within the cochlea was the same as in the preceding cases, except that there were none in the interscalar or modiolar vessels or in the perineural spaces of the cochlear fibers in the lamina spiralis ossea.

On both sides granules extended from the subarachnoid space through a circumscribed area of the adjacent layer of the dura into the perisaccular tissue of the medial inner wall of the endolymphatic sac (Fig. 1); some of them were also seen in the lumen of blood vessels, but none in the lumen of the sac.

Rabbit 128. Granules were found at the typical places in the scala tympani of the first and second turn. They extended along the cochlear fibers in the lamina spiralis ossea into the spiral ganglion, but not into the interscalar and modiolar vessels. Some granules were found only on one side in the scala vestibuli of the first turn. The rest of the scala was clear. In addition, granules had penetrated one side along the fibers of the utricular nerve into the stroma of the macula utriculi.

Rabbits 125 *and* 134. Prussian blue granules had penetrated on one side only into the scala tympani of the first and the adjacent parts of the second turn. They showed the usual distribution, but none of them could be seen in the perineural spaces of the cochlear fibers or in the interscalar or modiolar vessels. Very few granules were observed in the cochlear aquaeduct of the other side.

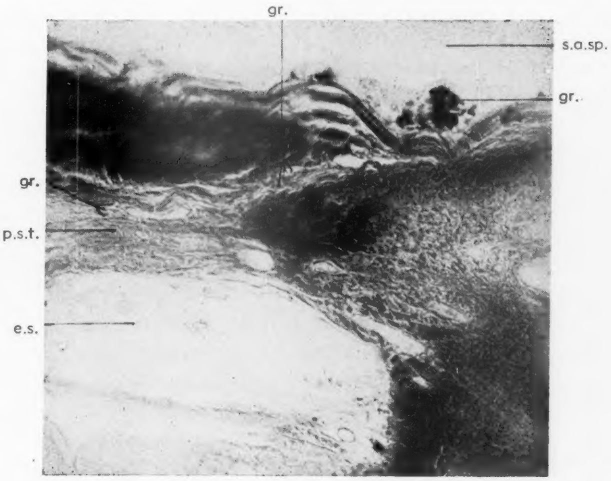

Fig. 1.—Rabbit 126. Granules accumulated in a circumscribed area of the subarachnoid space and extending from there into the perisaccular tissue. *e.s.*, endolymphatic sac; *gr.*, granules; *p.s.t.*, perisaccular tissue; *s.a.sp.*, subarachnoid space.

Rabbit 131. Granules had penetrated into the scala tympani of the first and the adjacent parts of the second turn and from there into the spiral ligament. No granules were seen, however, in Corti's organ or the prominentia spiralis, but a few in the crista spiralis. Granules were found in the dura covering the endolymphatic sac and in the subepithelial tissue of the medial wall of the proximal portion of the endolymphatic sac. No granules were present in the epithelial cells or in the lumen.

Comment: A review of the findings shows that in five cases the iron salts had penetrated into the cochlea on both sides, although not always to the same extent, and in two of them on one side only. There is no definite relationship between the extent of penetration into the cochlea and the time elapsed since the injection of the solution into the subarachnoid space or the amount of the injected fluid. The considerable variations in the results obtained can be explained by the differences in the structure of the reticular tissue within the aquaeduct and perhaps also by variations in the extent to which a limiting membrane, separating the reticular tissue from the lumen of the scala tympani, is formed. One must keep in mind, furthermore, that the solution was not entirely injected into the

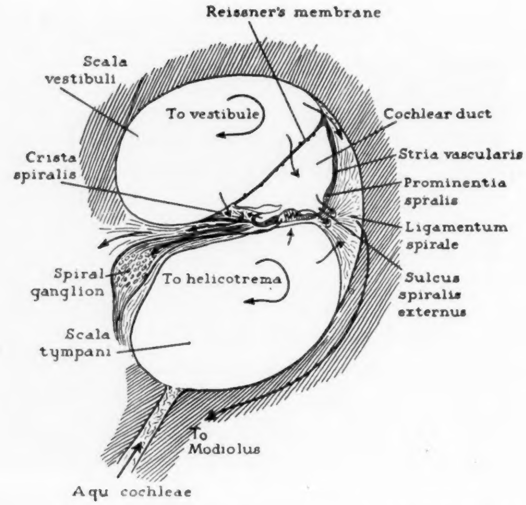
Reissner's membrane
Scala vestibuli
To vestibule
Cochlear duct
Crista spiralis
Stria vascularis
Prominentia spralis
Ligamentum spirale
Spiral ganglion
To helicotrema
Sulcus spiralis externus
Scala tympani
To Modiolus
Aqu cochleae

Fig. 2.—Diagram showing the movements of the salts after they have entered the scala tympani through the aquaeductus cochleae.

subarachnoid space. Sometimes a certain portion of it was lost by back-flow due to the movements or coughing of the animal.

Since, after injection of 0.5—0.75 cc of a 1 per cent solution, the salts could be detected in six minutes in the cochlea in only one rabbit out of five, and this to a very limited degree, and since after injection of 0.2-0.5 cc of a 5 per cent solution the salts were found in the cochlea in five rabbits out of seven, the most important factor in the speed of penetration would seem to be the concentration of the solution.

The distribution of the granules within the various structures of the cochlea indicates a typical pattern for spread of the salts (Fig. 2). Once they have entered the scala tympani they can be traced upward to the helicotrema and from there downward into the scala vestibuli. Only very small amount of the salts get, within 15 minutes, into the perilymphatic spaces of the vestibule or of the semicircular canals (rabbit 130). The granules found on one side in the perilymphatic cysterna of the vestibule after one and a half minutes (rabbit 127) are most probably evidence of a penetration of the fluid from the fundus of the internal auditory meatus.

Only a part of the iron salts which enter the scala tympani passes upward towards the helicotrema; another portion goes laterally into the stroma of the lower part of the spiral ligament and from there into the small veins within the latter. Still another portion goes between the fibers of the basilar membrane into the cochlear duct and into the various spaces between the cells of the organ of Corti. From there, the salts can be traced either in a lateral direction into the spiral ligament, mostly through the lower parts of the prominentia spiralis and the sulcus spiralis externus, or medially into the tissue and the small venous spaces of the crista spiralis or through the perineural spaces of the cochlear fibers into the spiral ganglion. From the spiral ligament the iron salts can be followed, sometimes within, but mostly along the somewhat larger vessels through the interscalar septa into the modiolus. In none of the specimens could blue granules be found in the vein which accompanies the cochlear aquaeduct. From the crista spiralus they could be traced in the lumen of the veins into the modiolus. Nearly all the salts which have entered the scala vestibuli through the helicotrema penetrate either through Reissners's membrane into the cochlear duct or into the upper most part of the spiral ligament or into the crista spiralis. In the seven specimens examined only a very small part of them had reached within fifteen minutes the perilymphatic spaces of the vestibule. Here, they can be traced into the lumen of trabecular vessels. All the salts which have entered the cochlear duct within the same period of time are resorbed there and none is passed through the reunient duct into the saccule or beyond. The blue granules found in the medial wall of the endolymphatic sac in five cases (rabbits 120, 121, 122, 126 and 131) are most probably the result of an intravital penetration from the subarachnoid space through the adjacent dural wall.

In those cases in which only smaller amounts of the injected iron salts have entered the scala tympani, they are resorbed either into the spiral ligament on the lateral wall alone or also through the basilar membrane into the cochlear duct of the first and successive portions of the second turn. If a large amount of iron salts has entered the scala tympani, not all can be resorbed from there; the rest extends into the scala vestibuli where additional resorption takes place. The findings in the seven cases indicate that the resorptive mechanism within the cochlea is highly effective and able to cope with even relatively large amounts of iron salts. The penetration from the fundus of the internal auditory meatus along the perineural spaces of the cochlear nerve into the spiral ganglion of the basal turn and into the stroma of the maculae and cristae is always insignificant in comparison with the penetration through the cochlear aquaeduct.

The findings in these rabbits confirm to a large extent the observations reported in our previous paper. An important new finding seems to be the demonstration of a spread of the salts from the subarachnoid space through a circumscribed area of the dura into the tissue of the endolymphatic sac and from there into some of the blood vessels. Granules found in this area, and even in the lumen of the sac, are not, therefore, as previously assumed, conclusive evidence of a current directed from the cochlea into the sac. The penetration of granules from the perilymphatic spaces into the lumen of some of the trabecular vessels deserves great interest because it indicates resorption of the perilymph in these areas. Furthermore, it was demonstrated in the specimens that the spread of the salts through the interscalar septa into the modiolus occurs mainly not within the blood vessels but in the perivascular spaces along the vessels.

EXPERIMENTS ON CATS

In a second series of experiments 0.1 cc of a 1 per cent solution of potassium ferrocyanide and iron-ammonium citrate was injected in four cats with a fine short bevelled needle directly into the scala tympani through the round window membrane after exposure and opening of the bulla. The injections were made on both sides, but a few minutes apart. The animals were killed five to sixteen minutes after the injection (see table 2) and fixed in 10% Formalin solution to which 5% hydrochloric acid was added.

The histological examination of the four cases showed the following:

Cat 601, *right side*. Both scalae of all turns contained Prussian blue granules. In the scala tympani accumulations of granules were seen on the surface of the spiral ligament and on the undersurface of the basilar membrane; in the scala vestibuli they were seen on the surface of the spiral ligament and of the crista spiralis and on the vestibular surface of Reissner's membrane a few granules scattered throughout its entire length from cochlear to cranial opening. In the basal turn granules were found in the stroma and some of the vessels of the spiral ligament bordering the scala tympani; within the cochlear duct, particularly in the spaces between the cells of Corti's organ; within the stroma and some vessels of the crista spiralis; around the prominentia spiralis; but to a very small extent in the stria vascularis. From the spiral ligament the granules could be traced in the perivascular spaces or in the lumen of some vessels through the interscalar septa into the modiolus, from the crista spiralis in and around some vessels into the modiolus, from the organ

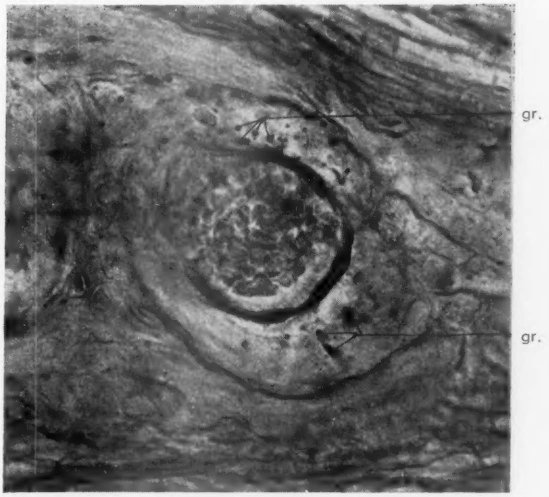

Fig. 3.—Cat 602. Granules (gr) in the perisaccular space of a modiolar vessel.

of Corti along the perineural spaces of the cochlear nerve fibers into the spiral ganglion. The changes were more marked in the lower than in the upper half of the basal turn. Above that they disappeared completely.

There were very few blue granules in the perilymphatic spaces of the vestibule and none in the semicircular canals.

The sacculus contained no blue granules. Some were seen in the saccular duct, but none in the utricle, the membranous canals, the endolymphatic duct, or the sac.

Left side. There were granules in the scala tympani of the first and second turn, but none above or in the scala vestibuli. A few scattered granules were found in the inner half of the cochlear aquaeduct, outward from the cochlear opening. The resorptive changes were of considerably lesser intensity, and again confined to the first turn. No granules were found in the vessels of the interscalar septa or in the lamina spiralis ossea. The perilymphatic spaces of the vestibule and the semicircular canals were free of granules. There were no blue granules in the saccule, but a few in the lumen of the proximal part of the endolymphatic duct. There were none in any other part of the peri- or endolymphatic system.

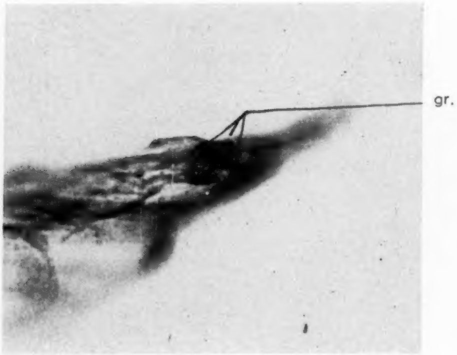

Fig. 4.—Cat 602. Granules (gr) within a small trabecular vessel in the vestibule.

Cat 602, *right side.* The scala tympani contained a moderate amount of blood in the first turn and a small amount in the second turn. Numerous granules were found in both scalae of all turns in the same distribution as in the preceding case. In addition, some granules were actually seen in the substance of Reissner's membrane and on its inner surface. The aquaeductus cochleae contained blood and blue granules throughout its entire length. Granules were also seen emerging from the cranial opening into the subarachnoid space.

The resorptive changes within the cochlea were very marked and found in all turns. From the spiral ligament some granules could be traced in the lumen, but most of these in the perivascular spaces of numerous vessels through the interscalar septa into the modiolus (Fig. 3) and from there in lesser numbers into the fundus of the internal auditory meatus. From the organ of Corti the granules could be followed along the perineural spaces of the cochlear nerve fibers into the spiral ganglion and from there along the perineural spaces and in the lumen of some small vessels to the trunk of the cochlear nerve and into the meshes of the arachnoid tissue in the fundus of the internal auditory meatus.

There were granules in the perilymphatic spaces of the vestibule and of the semicircular canals, sometimes also in the lumen of small blood vessels within the trabecula (Fig. 4) and on the outer surface of the saccule, utricle, and the membranous canals. Other granules were found within the lumen of the saccule and utricle, within the otolithic membranes, the epithelium, and in the subepithelial layers of the stroma of the maculae. From there they could be traced in some places along the perineural spaces of the saccular and utricular nerves into Scarpa's ganglion. Some were found in the endolymphatic duct and in the proximal, intraosseal part of the sac, but none in the intradural part. There were no granules in the lumen of the membranous semicircular canals, but some were present in the subepthelial layers of the stroma of the cristae ampullares.

Left side: A large tear was seen in the round window membrane closed by a blood clot. Both scalae of all turns contained more blood than those of the right side. The distribution of the blue granules and the extent and intensity of the resorptive changes were approximately the same as on the other side.

Cat 603. Both scalae of all turns contained many blue granules in the usual distribution. Intensive resorptive processes had taken place in all turns. There was a large accumulation of blue granules in the lowest part of the scala tympani, and extending from there throughout the cochlear aquaeduct up to but not beyond its cranial opening.

There was some fresh blood together with numerous blue granules in the vestibule and less numerous granules in the perilymphatic spaces of the semicircular canals. The saccule contained a small amount of fresh blood and numerous granules. The otolithic membrane and the epithelium contained no blue granules. There were granules within the stroma of the macula, particularly the basal parts, but none could be traced from there along the nerve fibers into the internal auditory meatus. The utricle contained none within the lumen. There were, however, numerous blue granules accumulated in the perilymphatic space on the outer surface of the wall of the macula and around the short stretch of the utricular nerve which traverses the perilymphatic space. They could be traced from there into the stroma of the macula. There were very few blue granules within the epithelium and none within the otolithic membrane. Granules also extended along the fibers of the utricular nerve, particularly in its peripheral parts, into the vestibular ganglion but not beyond it.

There were some granules in the lumen of the endolymphatic duct, more in the endolymphatic sac, particularly its proximal part. The sac contained, in addition, fine granular and homogenous pink-staining masses, polymorphonuclear leucocytes, small and large round cells, and some red blood cells. There were no blue granules within the epithelium and the perisaccular tissue.

There were no blue granules within the lumen of the semicircular canals, within the cupulae, or the epithelium of the cristae. They had accumulated, however, around the outer wall of the ampullae and the nerves entering the cristae, particularly the crista of the lateral canal. They could also be traced in the peripheral parts of the nerve into the stroma of the cristae and became more numerous along the lateral ampullar nerve into Scarpa's ganglion. The stroma of the two other cristae contained less granules; none could be observed in the two ampullar nerves towards the ganglion.

Left side. The cochlea contained only a small amount of granules in both scalae of all turns and some fresh blood in the scala tympani, particularly of the basal turn. Resorptive changes were found in all turns but of lesser intensity. The granules did not reach the modiolus, but extended only into the spiral ganglion. The aquaeductus cochleae again contained granules throughout its entire length.

There were only a few granules in the vestibule, mostly on the outer surface of the macula utriculi and around the trabeculae of the perilymphatic tissue. There were very few granules in the perilymphatic spaces of the semicircular canals. The lumen of the saccule was free from granules as were the endolymphatic duct and sac, the utricle, and the semicircular canals. Some fresh blood was seen in the lumen of the utricle extending into the ampulla of the external semicircular canal.

Cat 604, *right side.* There was a large tear in the round window membrane closed with a clot of blood. The scala tympani of the basal turn was completely filled with blood which, in the upper half of the turn, was increasingly mixed with blue granules. Some blood was found in the lower basal turn of the cochlear duct also. All the other parts of the cochlea were free from blood. Both scalae contained numerous blue granules in all turns and in the usual distribution. Extensive resorptive changes were seen in all turns. The acquaeductus cochleae was free from granules but contained some blood.

There were numerous blue granules in the perilymphatic spaces of the vestibule, particularly in the neighborhood of the macula utriculi. At the place where the vestibular vessels emerge from the bony wall, a small amount of fresh blood was seen. Numerous granules could be traced from the vestibule into the perivascular spaces of these vessels toward the fundus of the internal auditory meatus. Relatively few granules were seen in the lumen of some of the vessels. Frequently, granules were seen on the outer surface of the wall of the utricle, saccule, and the semicircular canals. In many sections it seemed that granules could be traced into the stroma of the walls.

Numerous blue granules were found in the lumen of the saccule, on the surface of the poorly preserved otolithic membrane, within the epithelium, in the stroma, and along the nerves leading from there to the vestibular ganglion either in the perineural spaces or in the lumen of some small vessels. There were blue granules in the peripheral parts of the stroma of the macula utriculi, but none within the epithelium, the otolithic membrane, or the lumen. The granules were not too numerous along the fibers of the utricular nerve, being found mostly in the lumen of small vessels. There were only a few blue granules in the arachnoideal meshwork in the fundus of the internal auditory meatus.

The endolymphatic duct and sac did not contain blue granules

There were some granules in the perilymphatic spaces of the semicircular canals. There was a greater amount in the stroma of the crista of the posterior ampulla and along the fibers of the ampullar nerve, but they were less numerous again in the cristae ampullares of the other canals. There were no blue granules in the lumen of any of the canals. The ampullae were collapsed and the cupulae had disappeared.

Left side. The tear in the round window membrane was smaller than on the right side. There were numerous blue granules and a small amount of blood in all turns of the cochlea. The membrane of Reissner was torn in some places and collapsed in all turns. Resorptive changes had taken place to approximately the same extent but a lesser degree than on the other side. The aquaeductus cochleae contained some granules throughout its entire length up to the cranial opening.

The perilymphatic space of the vestibule contained many granules which were particularly numerous on the outer surface of the wall of the macula utriculi and around the part of the utricular nerve traversing the lymphatic space. There also numerous granules in the perilymphatic spaces of the semicircular canals, some of which seemed to penetrate the wall of the membranous canals.

The wall of the saccule was extensively torn. The otolithic membrane was very poorly preserved and showed some granules on its surface; others were seen within the epithelium of the macula. More were seen diffusely distributed throughout the stroma and could be traced from there along the fibers of the macular nerve into the vestibular ganglion. The lumen of the utricle contained a moderate amount of granules; others were found in the otolithic membrane and the macular epithelium. They increased in number in the stroma, particularly in the peripheral parts, and could also be traced along the fibers of the utricular nerve into the vestibular ganglion; the majority of them lay in the lumen of small vessels within the nerve. Other granules extended from the vestibule into the perivascular spaces of the vessels which run from the fundus of the internal auditory meatus to the vestibule. Some were also found in the lumen of some of the vessels. Numerous granules were found in the arachnoid meshwork of the fundus of the internal auditory meatus.

There were numerous blue granules in the connective tissue surrounding the narrow portion of the endolymphatic duct, but none in the lumen of the duct itself or in the sac.

In all the semicircular canals the ampullae were collapsed and the cupulae had disappeared. There were numerous granules in the stroma of the three cristae which could be traced along the respective nerves into the vestibular ganglion and the arachnoid meshwork of the fundus. In the lateral and superior vertical cristae there were less numerous granules in the epithelium and the lumen of the adjacent areas of the canals.

COMMENT: It is not possible to determine accurately in each instance how much of the solution had actually entered the scala tympani, because frequently a certain part of the injected fluid flowed back into the middle ear, particularly when larger tears were produced in the round window membrane. Nevertheless, it can safely be assumed that in every case a far larger amount of the solution had entered the scala tympani than in any of the rabbits, injected intracystermally, and that a heavy overloading of the resorptive mechanisms of the cochlea had been effected. In most instances a varying amount of blood, resulting from the trauma of the injection, was found in the scala tympani and sometimes extended from there through the helicotrema into the scala vestibuli, the vestibule, and the perilymphatic spaces of the semicircular canals. Not all the blood, however, in the latter two areas must come from the scala tympani. The iron solution could have had a toxic effect upon the labyrinthine tissues and could easily have caused small hemorrhages such as took place into the right saccule of cat 603.

No definite relationship could be established between the degree and extent of the labyrinthine changes and the length of time elapsed since the injection because of the variations in the amount of fluid which had entered the cochlea in each instance and, perhaps, other yet-undetermined factors.

The iron salts injected into the scala tympani spread in four different directions. One part went into the aquaeductus cochleae and might have emerged from there into the subarachnoid space. This "reverse-flow" through the aquaeduct was seen in all but one instance, and in the latter (cat 604, right side) the lack of penetration was probably due to blockage of the aquaeduct by masses of blood. It is difficult to decide whether this reverse-flow is the manifestation of a simple diffusion of the salts or the result of an increase of pressure within the scala during the injection which forces the fluid in this direction.

The major portion of the injected fluid, however, spreads as in the rabbits injected cystermally along one of the following three ways: 1. from the scala tympani directly into the spiral ligament; 2. through the basilar membrane into the cochlear duct and from there (a) mainly through the prominentia spiralis and the sulcus spiralis externus but not through the stria vascularis into the spiral ligament, (b) into the limbus spiralis, (c) into the perineural spaces of the cochlear nerve fibers; 3. upward towards the helicotrema. On the upward movement a part of the salts is again resorbed into the spiral ligament or the cochlear duct of the upper turns, only the part which could not be disposed of in the scala tympani seems to reach the scala vestibuli. Here the iron salts spread downward, but on their way are again subject to resorption either through the spiral ligament, the limbus spiralis, or through the membrane of Reissner into the cochlear duct and from there again into the spiral ligament or the limbus spiralis. As a rule only small amounts reach the perilymphatic cysterna of the vestibule, a fact which again demonstrates the effectiveness of the resorptive mechanism of the cichlea.

From the spiral ligament the injected fluid spreads mainly in the perivascular spaces and to a much lesser extent in the lumen of some of the vessels, through the interscalar septa into the modiolus, and from there sometimes into the arachnoid spaces of the fundus or the internal auditory meatus. From the limbus spiralis the salts spread through the lamina spiralis ossea, either along or in the vessels, into the modiolus, from the organ of Corti in the perineural spaces of the cochlear fibers or in the lumen of small vessels into the spiral ganglion, and from there either into the trunk of the cochlear nerve or into the arachnoid spaces of the fundus. The vein accompanying the cochlear aquaeduct is again always free from granules.

Only a small amount of the iron salts which have passed from the scalae into the cochlear duct is not resorbed from the duct, and enters the saccule through the reunient duct.

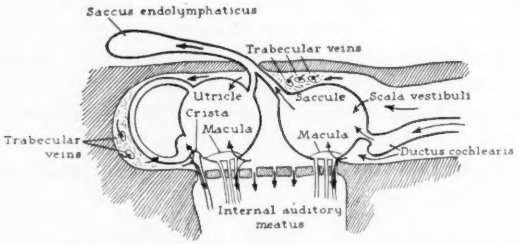

Fig. 5.—Diagram showing the movements of the salts after they have entered the vestibule through the scala vestibuli and the cochlear duct.

The interpretation of the fate of the salts which have entered the vestibule or the semicircular canals is difficult because of the simultaneous presence of two different kinds of changes which show considerable individual variations in their extent.

The iron salts which have penetrated into the perilymphatic spaces of the vestibule from the scala vestibuli spread from there into the perilymphatic spaces of the semicircular canal (Fig. 5). They enter the lumen of some of the small veins in the trabeculae of the perilymphatic tissue or penetrate, mainly along the perivascular spaces, through the inner wall of the vestibule into the arachnoid spaces of the fundus of the internal auditory meatus (cat 604); they also spread into the tissue surrounding the proximal part of the endolymphatic duct (cat 604, left side). Within the perilymphatic spaces themselves, larger accumulations of blue granules were found, particularly on the outer surface of the walls of the maculae of the saccule and utricle, and of the cristae ampullares, and also around the nerves which run from the vestibular or ampullar wall to these structures. These accumulations indicate the presence of movements of the salts towards these areas. The extension of granules from these locations along the nerve fibers into the stroma of the maculae, and to a lesser extent also of the cristae and also in the opposite direction, along the nerve fibers and sometimes in the lumen of small vessels towards Scarpa's ganglion and the arachnoid meshwork of the fundus, indicates a subsequent spread into two different directions. In some instances, such as in cat 602, the salts have evidently passed from the stroma of the maculae and cristae into the lumen of the endolymphatic spaces. There is also some evidence of a penetration of the salts through other parts of the walls of the membranous labyrinth into the lumen.

The salts which entered the saccule could be traced from there through the saccular duct (cat 602, right side) into the endolymphatic duct and sac, but in none of the cases was a large number of blue granules found in these cavities and in none was there evidence of resorption of the iron salts from the sac into the perisaccular tissue within 16 minutes after injection into the scala tympani. The possibility that a certain part of the salts is resorbed in certain areas from the lumen of the utricle, saccule, and ampullae into the stroma and from there along the nerves further outward must be considered, although the histological findings in the examined cases do not permit any conclusions. The granules in the lumen of the utricule and the semicircular canals seem to have come exclusively from the perilymphatic spaces by way of penetration along the nerve fibers at the indicated areas or directly through the membranous walls. There is nothing in the distribution of the granules which could be taken as an indication of a movement of the salts from these parts towards the endolymphatic sac.

EXPERIMENTS IN MONKEYS

In a third series of experiments Rhesus monkeys were studied. Varying amounts of spinal fluid were withdrawn by way of suboccipital cysternal puncture and replaced by the same or a slightly smaller amount of a 1-5 per cent iron salts solution. The animals were killed after three to twenty minutes. For details see Table 3.

The subarachnoid space contained, in all but two of the monkeys, less nmerous granules than in the rabbits. In some instances blue granules were also found in the fundi of the internal auditory meatus.

In one instance only were granules found within the cochlear aquaeducts and within the labyrinthine cavities (monkey 304).

The histological examination of this monkey showed the following:

There were numerous granules in the subarachnoid space extending into the fundi of both internal auditory meatus and from there along the fibers of the cochlear nerve for a short distance into the modiolus but not up to the spiral ganglion. Other granules could be traced in the perineural space of the saccular nerves into the stroma of the maculae sacculi. The epithelium and the otolithic membranes, however, were free from granules. On the right side they had also penetrated into the subepithelial tissue of the macula utriculi along the fibers of the utricular nerve. There weer no blue granules in the scala tympani, vestibuli, nor in the perilymphatic spaces of the vestibule or of the semicircular canals.

There were numerous granules in the spiral ligament and some within the lumen of the cochlear duct of all turns with exception of the lowest part of the basal turn. They were particularly numerous in the tissue spaces and the lumina of the vessels and in the epithelium of the sulcus spiralis externus and the prominentia spiralis; they were also found in the adjacent parts of the spiral ligament, but there were hardly any in the other parts of the ligament. Some granules were present in the tissue spaces of the crista spiralis and in the lumen of some small vessels in this area. There were some within the various spaces of Corti's organ, but few in other parts of the lumen of the cochlear duct. There were none within the lamina spiralis ossea.

The cochlear aquaeducts were long and very narrow. The right aquaeduct contained granules within the meshes of the reticular tissue in its outer four-fifths, the left one in its outer two-thirds. The innermost parts of both aquaducts contained no blue granules.

The utricle, saccule, the membranous semicircular canals, and the proximal part of the endolymphatic duct had no blue granules.

Numerous granules were present in the wall of the distal part of the duct and the rugose portion of the endolymphatic sac becoming less numerous in the distal part of the sac. The tissue spaces and the lumen of the small blood vessels of the perisaccular tissue contained granules, with lesser numbers in the lining epithelium and the lumen of the sac and duct. Granules were also found in some of the free cells within the lumen. Towards the distal part of the sac the granules within the tissue spaces were less numerous; the blood vessels were somewhat larger in this area and densely filled with granules. These were, as a rule, more numerous in the part of the sac away from the subarachnoid space than in the part adjacent to it. There were few granules within the medial leaf of the dura which separates the duct and sac from the subarachnoid space.

The granules were, in general, more numerous on the right than on the left side.

Comment: The greater length and the narrowness of the aquaeductus cochleae in monkeys seems mainly responsible for the fact that in only one case out of nine were granules observed within the cochlea. In this latter case, the presence of granules in the fundi of the internal auditory meatus, in the perineural spaces of the cochlear nerves, the saccular and the right vestibular nerves, and the respective maculae, but not in the epithelium and the lumina of the saccule or utricle indicates a movement of the salts from the internal auditory meatus into the stroma of the maculae but not beyond these areas. The presence of granules in the cochlear aquaeducts demonstrates a spread from the subarachnoid space towards the scala tympani. This assumption derives additional support from the fact that granules of precipitate were found in the cochlea only in the one case in which they were also present in the aquaeduct; they were more numerous in the cochlear duct and the cochlear aquaeduct of the same side. One must assume that practically all iron salts have already passed from the scalae into the cochlear duct and are in the process of resorption from there. In view of the findings in rabbits where the resorption from the scalae and the cochlear duct was already in full swing after one and a half minutes (rabbit 127) such an assumption is not unreasonable. The resorption from the cochlear duct seems to take place in monkeys in the same place as in rabbits and cats.

The absence of granules within the lowest part of the cochlear duct, the saccule, and utricle suggests that the numerous granules found in the connective tissue surrounding the endolymphatic sac are not connected with those present in the cochlea. It would be hardly conceivable that within nine minutes all these iron salts should be carried through an endolymphatic current all the way down from the cochlear duct without leaving any traces in the intervening parts. penetration of the solution from the subarachnoid space through the dura into the perisaccular tissue and the blood vessels and to a limited extent into the lumen, similar to the movement of salts described in the rabbits, seems more probable. It can, however, not be proved conclusively because no granules were found in the medial leaf of the dura, but there were granules in the perisaccular tissue of the lateral wall which is farther away from the dura than the medial wall. Since a hematogenous passage of the iron salts seems entirely out of question, penetration from the subarachnoid space remains, nevertheless, the only possibility to be seriously considered.

One can, therefore, say that as far as the findings in one specimen permit any conclusion at all, the salts seem to have penetrated from the subarachnoid space in three directions, through the cochlear aquaeduct into the cochlea, through the internal auditory meatus into the macula sacculi and utriculi, and through a circumscribed area of the dura into the loose perisaccular tissue and eventually into the lumen of the sac. Further investigations will show whether these assumptions are actually correct.

DISCUSSION

In the experiments described in this paper the distribution of Prussian blue granules within the cochlea was studied after injection of solutions of potassium ferrocyanide and iron-ammonium citrate into the cerebellomedullar cysterna of rabbits and monkeys, and into the scala tympani of cats; it followed essentially the same pattern in the three species.

The spread of the injected iron salts from the subarachnoid space into the fundus of the internal auditory meatus and the cochlear aquaeduct could be the result of the currents within the cerebrospinal fluid and of the pulsation of the intracranial vessels together with the action of all the physico-chemical factors governing the distribution of dissolved substances within fluids. It does not necessarily require the assumption of a special fluid current directed from the subarachnoid space towards the inner ear. Biological membranes encountered by the dissolved salts could modify but not materially change the spread towards the inner ear.

A variety of physico-chemical factors might explain why Gisselson[5] found that 0.5 cc of a 20 per cent solution of fluorescein sodium injected into the subarachnoid space of cats did not appear in the perilymphatic space before one hour after injection. After intravenous injection of 1.0-1.5 cc of a 10 per cent solution, fluorescein pases into the cerebrospinal fluid after a few minutes, but, evidently due to the smaller concentration in the latter, it takes three hours before it passes into the perilymphatic fluid through the cochlear aquaeduct. In guinea pigs it does not appear at all in the perilymphatic space even four hours after intravenous injection of 0.5-1.0 cc of a 10 per cent solution. There seems, however, no essential difference between Gisselson's and our findings.

A fluid current from the subarachnoid space to the inner ear has, however, been deduced from the fact that insoluble substances such as cinnabar, India ink, animal charcoal, oxalated chicken blood, or an emulsion of olive oil (Nakamura)[12] introduced into the subarachnoid space might also penetrate into the inner ear (see Altmann and Waltner,[2] 1947). Sometimes, in man, blood is found in the scala tympani following a hemorrhage into the subarachnoid space, again regarded as suggestive of a current in the same direction. A spread of these substances into the aquaeductus cochleae, which is in a certain sense only a recess of the subarachnoid space, can, however, not be taken as convincing proof for the existence of such a fluid current. Since the particles in question are very small, their spread into all parts of the subarachnoid space, their penetration into the scala tympani, and their passage from the fundus of the internal auditory meatus into the modiolus or other parts of the inner ear could again be the result of the physico-chemical factors governing the distribution of small particles. As already pointed out in our previous paper, the degree of development of a limiting membrane between the reticular tissue of the aquaeduct and the lumen of the scala tympani shows variations in animals; there is no continuous membrane demonstrable in every section. Penetration into the scala could, therefore, take place through areas in which the membrane was not well developed. Another possibility to be considered would be that during the injection of the solution into the subarachnoid space a sudden increase of the intracranial pressure had occurred, which could have caused tears in the limiting membrane and thus have facilitated the passage of particles into the scala tympani.

It seems, therefore, more probable to assume that there is only very little, if any, fluid exchange at all between the subarachnoid space and the labyrinthine cavities. This view has also been expressed by Alfred, Hallpike, and Ledoux[1] who found the osmotic pressure of the labyrinthine fluids in the cat higher than that of the cerebrospinal fluid, an observation which was confirmed by Ledoux.[8,9] This author also found the index of refraction of the labyrinthine fluids in dogs and cats higher than that of the cerebrospinal fluid, another proof for the relative independence of the two from each other. The morphological changes after occlusion of the cochlear aquaeduct vary and are interpreted differently by different authors (Wittmaack,[22] Karlefors,[7] Uyama,[19] Takahara,[17] Ohma,[13] Waltner[20]).

The possibility that a certain small proportion of the perilymph originates from the cerebrospinal fluid and enters via the cochlear aquaeduct and perhaps also through the internal auditory meatus cannot be definitely denied, but the cerebrospinal fluid does not seem an essential source of the perilymph. The assumption of a regular fluid current from the scala tympani to the subarachnoid space is not substantiated by the experimental findings. A penetration of dissolved substances in this direction might take place, however, if their concentration in the scala tympani was very great as compared with that in the subarachnoid space, as in our cats.

The distribution of the iron salts which have entered the scala tympani within the various structures of the cochlea cannot be explained solely by the action of physico-chemical factors. The accumulation of granules in certain areas of the spiral ligament and in the crista spiralis and the relative freedom from granules of other areas, such as the stria vascularis, indicates the action of specific biological factors. It points to a resorption of the iron salts in certain areas into the tissue spaces. From there the salts can be traced into blood vessels or into the fluid spaces which surround the modiolar vessels and from the latter into the fundus of the internal auditory meatus. The main resorption of the iron salts seems to take place from the cochlear duct and a lesser amount only directly through the scalae. This is facilitated by the free permeability of the basilar membrane and Reissner's membrane for the salts. Since salts are only resorbed in dissolved form together with fluids, fluid movements must exist, directed in the scalae towards the lower and upper parts of the spiral ligament and in the cochlear duct towards the external spiral sulcus, the spiral ligament, and the crista spiralis. The spread of the salts within the scala tympani upward to the helicotrema, and from there downward through the scala vestibuli, is therefore, at least partly, controlled by the actual fluid movements within the scalae and by the fact that a large-scale penetration through the basilar membrane and Reissner's membrane takes place. The biological forces behind these fluid movements are similar to those controlling the production and resorption of the cerebrospinal fluid.

The direction of the fluid currents within the cochlear duct and the great intensity of the resorptive processes in certain parts of the latter might also explain why in Gisselson's experiments in cats the fluorescein entered the endolymph just as quickly as the cerebrospinal fluid, but did not penetrate from there into the perilymphatic space.

A partial confirmation of our views is furnished by the histologic findings of Saxen[15] according to whom desquamated and degenerated epithelial cells and other debris in the cochlear duct in dogs and man are removed through the resorptive action of the epithelium of the sulcus spiralis externus. The extent to which by the same way endolymphatic fluid is also removed could not be found out by the histologic methods employed by Saxen. That this resorption of fluid actually takes place is, however, proved by our experiments.

The conclusions arrived at by us, that a very active resorption takes place in the cochlear duct, do not contradict the existence of a fluid current within the cochlear duct directed towards the endolymphatic duct and sac as indicated by the experiments of Guild.[6]

The results of our experiments, however, seem to demonstrate quite clearly that the current from the cochlea to the endolymphatic sac is without great significance for the resorption of low molecular substances in physiological concentrations from the cochlea. The latter will be disposed of by the resorptive mechanism within the cochlear duct. Only when excessive amounts penetrate into the cochlear duct, as in Guild's guinea pigs, Yamakawa's[23] rabbits, or in our cats, will they be carried down to the sac. The metabolites resulting from the function of the cells of the organ of Corti which penetrate into the endolymph are evidently also removed by this local resorptive mechanism. From the teleological standpoint of body economy, it would be hard to understand why, for instance, metabolites originating in the apical turn of the cochlea should be carried all the way down through the cochlear duct into the saccule and thereby come in contact with other parts of the organ of Corti.

The resorption from the cochlear duct of high molecular substances, of emulsions of fats, and of corpuscular elements is perhaps less efficient than that of the low molecular substances used in our experiments. It is conceivable that larger proportions of these substances might reach the endolymphatic sac and be taken up here by phagocytes or resorbed through the wall; a similar opinion has been expressed by Mygind.[11] This view helps to explain the findings of Anderson[3] in intravitally stained guinea pigs. After protracted intravital staining with trypan-blue, two, three, and four days after the last injection of the dye the presence of trypan-blue could be demonstrated histologically in the form of blue granules in histiocytes in the perisaccular tissue of the endolymphatic sac in the so-called intrasaccular cells, and also as diffuse blue stain in the endolymph of the saccus; whereas no dye was found here seven days after the last injection. The rest of the internal elements of the labyrinth was always colorless. After a single injection (intraperitoneal or intravenous) it was possible in animals killed after a certain interval to demonstrate a diffuse coloring of the endolymph in the endolymphatic sac (the labyrinthine endolymph was always colorless).

It was further found by Anderson that staining of the intrasaccular cells appeared later than coloring of the endolymph in the sac. Anderson assumed from these findings that trypan-blue introduced into the organism passes into the endolymph, and that the intrasaccular cells absorb the dye from the endolymph. In contrast to the saccular endolymph, the labyrinthine endolymph was colorless in every specimen examined. This finding was confirmed by direct observation of the internal parts of the labyrinth immediately after death and also in vivo. The relative considerable accumulation of trypan-blue found in the saccus and limited thereto was explained by Anderson in the following way. The dye has passed in minute amounts from the blood stream over a large area of the labyrinth into the endolymphatic system, and the endolymph has transported it to the saccus where it has been retained for a time before being resorbed by th intrasaccular cells which then presumably emigrate through the saccular wall. Owing to conditions still unknown, the passage of the dye from the bloodstream to the endolymph takes place to a far lesser extent than from the blood stream to other tissues.

In our experiments that portion of the iron salts which was not resorbed from the scalae eventually reaches the vestibule, particularly when their concentration in the perilymph was great as after direct injection of iron salts into the scala tympani of the cats. In these instances, the iron salts had also spread from the scala tympani through the cochlear aquaeduct into the subarachnoid space. This would have been difficult to explain if a strong fluid current from the subarachnoid space into the scala tympani had really existed as assumed by many authors. It becomes, however, easily understandable if one assumes that there is no, or hardly any, fluid current within the cochlear aquaeduct and that salts can spread through the latter in either direction depending upon their relative concentration in the cerbrospinal fluid and in the labyrinthine fluids.

From the vestibule the iron salts penetrate into the perilymphatic spaces of the semicircular canals. They are also seen in small vessels in trabecula of the perilymphatic tissue, a finding which points to resorptive changes in these areas. The walls of the saccule, utricle, and of the semicircular canals are also to some extent permeable to the iron salts. Other portions of them spread along the nerve fibers into the stroma of the maculae and cristae and eventually into the lumen of the saccule, utricle, and of the ampullae. Still others can be traced through the medial wall of the vestibule into the fundus of the internal auditory meatus, either in perineural or in perivascular spaces. As in the trabecula of the perilymphatic tissue, the blue precipitates of the iron salts are also seen in the lumen of small vessels in the stroma of the maculae and cristae and within the branches of the vestibular nerve, a finding indicative of their resorption into the blood stream in those places with fluid movements directed to the resorptive areas.

The spread of salts from the vestibule to the fundus of the internal auditory meatus gives rise to the same questions as the spread from the scala tympani to the subarachnoid space and from the cochlea along the perivascular spaces of the modiolar vessels into the fundus. Since it would be incompatible with the existence of a strong fluid current from the fundus into the labyrinth, one comes again to the conclusion that such a current does not exist, and that a spread occurs in either direction depending upon the relative concentration of the salts in the fundus and the vestibule.

Nothing definite can be said about the main source of the perilymphatic fluid. It seems much more probable that it is derived from the perilymphatic blood vessels than that it originates from the endolymph and passes through the walls of the membranous labyrinth. According to Mygind, in the guinea pig the perilymph is presumably formed in a capillary network in the uppermost part of the spiral ligament just above the stria vascularis. The walls of the membranous labyrinth seem to be easily penetrated by low-molecular substances, and this fact could explain why the peri- and endolymph are virtually isosmotic. The fact that in our experiments the iron salts moved from the peri- into the endolymphatic space, simply demonstrates the high degree of permeability of the membranes and should not be taken as an indication that this is the prevailing direction of movement of dissolved substances under physiological conditions. Normally, the prevailing direction is more likely to be from the endolymph towards the perilymph.

The resorption of the cochlear perilymph seems to take place from both scalae into the tissue spaces and from there into the blood vessels of the spiral ligament; from the vestibule and the semicircular canals into the blood vessels of the trabecula of the perilymphatic tissue. The spread into the perivascular and perineural spaces is probably of lesser importance under physiological conditions. Both spaces are more likely to represent auxiliary avenues of escape for dissolved substances if their concentration is so high that the normal pathways have become overloaded.

The salts which have passed into the lumen of the saccule can be traced from there into the endolymphatic duct and sac. Because resorption of endolymph takes place in certain areas of the endolymphatic sac, the salts have probably been carried down to these areas not solely by diffusion but also by an actual fluid current. That no evidence of resorption from the lumen of the sac was seen in any of our specimens could have been due to the relatively short time interval between the injection of the salts and the death of the animals.

Great caution is advisable in the interpretation of the findings in and around the endolymphatic sac because of diffusion of the salts from a circumscribed area of the subarachnoid space along a projection of the dura into the perisaccular tissue. Here the presence of granules could easily be mistaken as evidence of resorption from the lumen or of transudation from the bloodstream. A current from the utricle to the endolymphatic sac was assumed by Doi[4] who found particles of India ink in the endolymphatic sac thirty minutes after injection into the lumen of the membranous canals. Increased pressure is, however, unaviodably present in such experiments. Siirala[16] came to the same conclusion as Doi on the basis of histologic studies of the sac. There was nothing in the distribution of the granules in our specimens which could be interpreted as an indication of such a current.

The possibility of a local resorption of endolymph from the lumen of the saccule and utricle into the stroma of the maculae and from the ampullae into the stroma of the cristae must be seriously considered, although the histologic findings in the examined cases do not permit any definite conclusions regarding these questions.

SUMMARY AND CONCLUSIONS

Using the method of Weed, iron salt solutions of various concentrations were injected into the subarachnoid space of 15 rabbits and 9 rhesus monkeys. The spread of the salts into the inner ear was studied after fixation of the specimens in formalin-hydrochloric acid solutions. Similarly, in 4 cats iron salts were injected through the round window membrane directly into the scala tympani.

In order to avoid misunderstanding in the interpretation of the findings, one must distinguish between spread due to actual fluid currents, due to factors such as diffusion and osmosis, and various other biological factors responsible for the resorptive changes occurring in certain well defined areas. One must furthermore keep in mind that resorption of salts takes place together with resorption of fluid, and that, therefore, fluid currents must be assumed, directed to the areas of resorption.

The iron salts spread readliy through the wide aquaeductus cochleae of rabbits and less readily through the narrow aquaeduct of monkeys into the scala tympani. Another less extensive spread may occur from the fundus of the internal auditory meatus mainly in the perineural spaces of the respective nerves into the modiolus, the maculae, and the cristae ampullares. Membranes encountered by the iron salts do not seem to slow up this spread perceptibly. In the cats, the iron salts spread in the opposite direction; from the scala tympani through the aquaeduct into the subarachnoid space, from the modiolus, the vestibule, and the perilymphatic spaces of the semicircular canals into the fundus of the internal auditory meatus.

These findings are regarded as an indication that active fluid currents are absent in either direction, and that the spread of the iron salts is mainly or entirely the result of the action of the usual physico-chemical factors.

In the scala tympani the salts spread partly upward to the helicotrema and partly through the basilar membrane into the cochlear duct, while another part was resorbed into the spiral ligament. From the helicotrema the salts spread in the scala vestibuli towards the vestibule. A part of them passed through Reissner's membrane into the cochlear duct, and another part was resorbed directly into the spiral ligament. If sufficient amounts of iron salts reach the vestibule, they spread from there into the perilymphatic spaces of the semicircular canals and eventually into the blood vessels of the trabecula of the perilymphatic tissue. Other portions pass through the walls of the membranous labyrinth into the endolymphatic space. If the concentration of the salts in the perilymph is very high, they may spread along the perineural and perivascular spaces in either direction towards the maculae and cristae and into the fundus of the internal auditory meatus. The findings indicate that actual resorption of perilymphatic fluid takes place mainly in three areas: from both scalae into the spiral ligament, and from the vestibule and the semicircular canals into the blood vessels in the trabecula of the perilymphatic tissue.

Since no fluid current towards the perilymphatic space could be demonstrated, either from the subarachnoid or from the endolymphatic space, at least the main part of the perilymphatic fluid is evidently formed in the perilymphatic space itself most likely from the blood vessels. The marked permeability of the walls of the endolymphatic system makes it probable that a spread of dissolved substances takes place from the endo- into the perilymphatic fluid or vice versa. This would signify that both are virtually isosmotic.

Within the cochlear duct resorption of iron salts occurs through the sulcus spiralis externus and the spiral ligament and through he crista spiralis, indicating the resorption of endolymph in the same areas. The resorptive mechanism of the cochlea seems more than sufficient for the disposal of low molecular substances in physiological concentrations. Although resorption of the endolymph from the saccule, utricle, and the ampullae of the semicircular canals seems probable, no definite evidence of it could be found in the specimens.

Resorption of the endolymph from a certain part of the endolymphatic sac, although not visible in our specimens, seems, however, an established fact and apparently indicates the presence of slow fluid current towards the sac. This current seems to affect mainly those substances which are difficult for resorption by the regular cochlear mechanism and are carried into the sac where they are broken up by the phagocytes and eventually resorbed. Presence of granules in the subepithelial tissue of the endolymphatic sac is, however, not necessarily an evidence of resorption from the lumen because of diffusion of the salts from a circumscribed area of the subarachnoid space along a projection of the dura into the perisaccular tissue.

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TABLE 1.—DETAILED DATA ON THE RABBIT EXPERIMENTS.

RABBIT NUMBER	CC'S OF SPINAL FLUID WITHDRAWN	CC'S OF SOLUTION INJECTED	PER CENT OF SOLUTION	MINUTES BETWEEN INJECTION AND KILLING
120	0.5	1.0	1	2
121	0.5	1.0	1	5
122	0.5	1.0	1	1
123	0.75	1.0	1	4.5
124	0.5	1.0	1	6
125	0.6	0.3	5	3
126	0.6	0.3	5	5
127	0.6	0.4	5	1.5
128	0.6	0.3	5	2.5
129	0.6	0.5	5	1.25
130	0.6	0.4	5	15
131	0.5	0.25	5	10
132	0.6	0.4	5	7.75
133	0.6	0.3	5	6

TABLE 2.—DETAILED DATA ON THE CAT EXPERIMENTS.

CAT NUMBER	CC'S OF SOLUTION INJECTED	MINUTES BETWEEN INJECTION AND KILLING	
		RIGHT	LEFT
601	0.1	12	6
602	0.1	7	5
603	0.1	7	10
604	0.1	14	16

TABLE 3.—DETAILED DATA ON THE MONKEY EXPERIMENTS.

MONKEY NUMBER	CC'S OF SPINAL FLUID WITHDRAWN	CC'S OF IRON SALT INJECTED	PER CENT OF SOLUTION	MINUTES BETWEEN INJECTION AND KILLING
301	0.7	0.5	5	12
302	1.0	0.8	5	20
303	1.0	0.8	5	12
304	1.0	0.8	5	9
305	1.0	0.8	5	6
306	0.25	0.25	5	3
307	0.7	0.6	1	15
308	0.9	0.7	1	10
309	0.8	0.6	1	6

LXIII

ESOPHAGEAL SPEECH

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In the treatment of cancer of the larynx the operation of total laryngectomy plays a major role. In these cases the problem is manifold: first and of greatest importance, naturally, is complete eradication of the growth and recovery from the operation. The next phase of the problem is that of speech re-education, the most important factor in the eventual rehabilitation of the patient, so that he may again become a reasonably contented, gainfully employed, and socially acceptable member of society.

There are several methods by which artificial speech may be produced, but it is generally agreed that the method known as esophageal speech, if it can be acquired, is preferable to either the reed larynx or the electrolarynx. Too often, however, the mechanism of esophageal speech has been presented to the patient and to the public, clothed in an air of mystery and compounded by the use of complicated and obscure verbiage.

As a matter of fact, esophageal speech differs from normal speech mainly in the anatomic structures which are involved. The basic form of the mechanism is the same; that is, a column of air is made to pass from a reservoir through a relatively narrow aperture—the edges of which being in contact—are made to vibrate and, thereby, to produce sound which entering the mouth is articulated by the tongue, lips, and teeth to produce speech. In normal speech the air from the reservoir of the lungs and the bronchial tree passes by way of the trachea through the narrow aperture of the glottis (vocal cords). After total laryngectomy the reservoir of air for voice production is in the esophagus (rarely the stomach) rather than in the tracheobronchial tree. When this column of air is expelled or eructed upward, it produces a vibration of soft tissues surrounding a relatively narrow aperture at about the level of the cricopharyngeus or, in some cases, at a higher level in the hypopharynx. When these soft tissues vibrate, a sound is produced which is identical with the belch in the normal person. If the laryngectomized

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person can belch—particularly if he is capable of a sustained belch—he can produce articulated esophageal speech.

Those who have made a study of this phenomenon are agreed upon its mechanism, but some observers often go to undue lengths to identify the neuromuscular structures (pharyngeal constrictors, supra- and infrahyoid muscles and their nerve supply) and the sequence of voluntary and involuntary motions which are involved in gulping air into the esophagus and then expelling it. Although such studies may be of some academic interest, in actual practice they tend to confuse and render more difficult the teaching and learning of a commonplace act, namely, the production of a belch.* McCall[1] was one of the first to emphatically define the mechanism of esophageal speech by its correct, though what, by some, might be considered an inelegant** and unscientific name. He has advised that before total laryngectomy the preoperative period "be utilized in learning to belch . . . " which "will greatly shorten the postoperative training for the development of esophageal voice." Whether or not such preoperative instruction is practical and psychologically suitable to most patients is not the point under discussion at this time. The purpose of this communication is rather to emphasize the homely and commonplace character of esophageal speech and to strip it of the encumbrance of mystery.

The time has passed when there need be any further enigma about the mechanism of esophageal speech. Unfortunately, there are those who claim to have "invented a new method" of esophageal speech. The only excuse for such misrepresentation is that some isolated laryngectomee acquires the art by himself, is therefore led to believe that he invented it, and in his enthusiasm innocently professes to be the first to have acquired the act and a new method of teaching it. As a matter of fact, esophageal speech, self-taught, is almost as old as laryngectomy itself. A case is reported in the literature[2] of a patient of Dr. J. Solis-Cohen of Philadelphia, a

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*Many physicians will doubtless recall the apocryphal story of the medical school instructor who included this question in a final examination in physiology: "Give the name of the following physiological act." And then came a lengthy description of a series of neuromuscular actions whose complicated verbiage might lead the unsuspecting to conclude that long practice and training would be necessary to carry out such an intricate act. The answer, however, could be stated with a single word: namely, sneeze. The same type of involved terminology is sometimes found in discussions of esophageal speech to describe the ordinary belch.

**It is inherent in occidental culture that the act of belching is considered offensive and indelicate and consequently, vulgar. For this reason, most discussions of esophageal speech do not include this necessarily descriptive term. On the other hand, by oriental custom the act of belching by the guest after a meal is considered a complimentary gesture to the excellence of the cuisine.

laryngectomee, who over a half century ago, in 1895, had himself developed esophageal speech so successfully that "he had no difficulty in making himself heard in a room that ordinarily held three hundred people."

Speech training following laryngectomy is probably best carried out in specially organized clinics by the group method of teaching, rather than by individual instruction. The instructor himself should be a laryngectomee whose special talents consist not only of an excellent esophageal voice, which may be used as a standard, but also the ability to inspire confidence that by persistent effort the laryngectomee can again acquire a spoken voice and return to a normal, gainful mode of life. There is no necessity for the complicated verbiage and pseudoerudite discussions which tend to obscure such simple, plain basic facts readily understandable to the unfortunate laryngectomee who is faced with the necessity of mastering some new method of speech. There is no need for the use of such a term as "buccopharyngeal speech" as being different from esophageal speech—actually a distinction without a difference. There is only one mechanism involved, namely, the eructation of air (from the esophagus and possibly the stomach) through a narrow aperture which lies somewhere in the hypopharynx. The exact level of these vibrating tissues may be anywhere from the level of the cricopharyngeus up to the level of the base of the tongue; in other words, wherever the healing of the tissues in the operative field has produced the narrowest aperture in the soft tissues which can be made to vibrate.

737 PARK AVENUE

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LXIV

FISHBONE IN MEDIASTINUM REMOVED BY ESOPHAGOSCOPY

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Every experienced endoscopist has records of sharply pointed objects which have perforated the esophageal wall and caused mediastinal infection. Morbid processes in more remote parts of the body, as a result of the intruder's ability to travel, have occasionally been encountered. Fatal hemorrhage from erosion of a great vessel has also occurred with surprising frequency.

Penetration of the esophageal wall may occur prior to esophagoscopy or following efforts to remove the foreign object. The time that has elapsed since the ingestion of the foreign body is not necessarily a criterion of the extent of the damage. A sharp, slender object, such as a fish bone, has often been known to penetrate entirely through the wall of the esophagus in a matter of hours. External operation to control the resulting infection and to recover the foreign body has been considered a grave and frequently fatal undertaking.

I recently was able to recover successfully by esophagoscopy a fish bone which had become lost in the mediastinal structures. This unique experience seems worthy of recording.

REPORT OF CASE

Mrs. N. G., age 58, summoned me to her home on September 4, 1948, on account of a choking episode following the swallowing of a bolus of meat. She related that 3 days previously, while eating fish, she had felt a bone lodge in her throat. A neighborhood physician whom she promptly consulted could not find the bone and expressed the opinion that her symptoms were due to the scratching effect of the bone on the mucous membrane of the pharynx and that the bone had probably descended into the stomach.

During the next three days she had experienced increasing difficulty with her meals but it was not until the recent choking spell on attempting to swallow the bolus of meat that she had become distressingly uncomfortable.

Examination of the throat and mirror examination of the hypopharynx and larynx revealed no signs of foreign body. A roentgenogram was made the following morning at St. Joseph's Hospital and a thin bone was found delineated on the film opposite the seventh cervical vertebra.

Esophagoscopy was performed one hour later, using ether anesthesia. A bolus of meat was found below the cricopharyngeal area, grasped with alligator forceps and removed. Careful search was then made for the fish bone but it could not be located anywhere in the esophagus. The bolus of meat was also carefully inspected but the bone could not be identified. An ecchymotic area, 2 mm in diameter, was seen on the right lateral esophageal wall just below the cricoid level and it was thought that this was the result of the trauma inflicted by the sharp edge of the bone. I presumed that the bone had become dislodged during the manipulation attending the grasping and extraction of the bolus of meat and that it had by now found its way into the stomach.

The patient was returned to her room and given sterile liquids and Duracillin 300,000 units every 24 hours for the following two days. Although she expressed herself as feeling improved, relief did not appear to be complete and I suspected that the bone might still be lodged in the esophagus. A check roentgenogram on September 7, 1948, revealed the bone to be approximately in the same location as in the previous film.

Esophagoscopy was repeated on September 8, 1948, and the bone could not be found. The ecchymotic area seen at the previous examination was still apparent and was the only visible clue as to the possible location of the bone. I then concluded that it had entirely penetrated through the esophageal wall at this point and had become completely lost in the adjacent structures. Using a delicate, forward grasping Jackson forceps, I gently insinuated it into the center of the ecchymotic area. The forceps seemed to enter a preformed channel to a depth of about 3 mm. I carefully opened the forceps and closed it with a grasping action. After several attempts I finally succeeded in extracting the bone (Fig. 1).

The patient made an uneventful recovery.

The fish bone in this case was, strictly speaking, no longer a foreign body in the esophagus, in that it had left the confines of the esophageal lumen. Success in its extraction by esophagoscopy was fortuitous and the method is not to be recommended for routine use as the dangers of penetrating the esophageal wall are too well known to require comment here. However, in this instance the

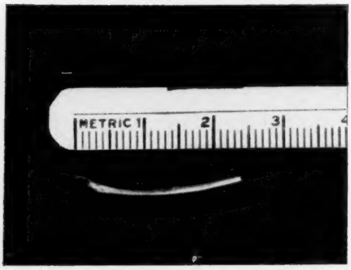

Fig. 1.—Fish bone 2.5 cm (1 inch) long extracted from the para-esophageal structures.

preformed channel made by the fish bone could be fairly accurately followed. I felt justified in exploring this channel for a few millimeters and was rewarded by recovering the intruder without causing apparent additional trauma.

That the bone would otherwise have ultimately set up a mediastinal infection requiring external drainage is obvious. Nor can the tendency of a pointed object, by advancing to erode into a large vessel, including the aorta, be lost sight of. This tragic outcome has been amply stressed in the recent literature.[1, 2, 3, 4]

In some cases reported, only a few hours or a few days have sufficed to bring about this fatal termination. An instance has recently been recorded in which a bone ultimately penetrated into the right renal artery.[5]

Even coins, after prolonged sojurn in the esophagus, have been known to erode into the aorta.[6]

The fact that this patient was spared the risks or even fatal outcome of a complicating mediastinitis, empyema, pyopericardium or erosion of a great vessel, seems worthy of recording. The method of access to the fishbone was certainly unique in my experience, nor have I found reference to a similar approach recorded in the recent literature which I have examined.

CONCLUSIONS

A case is reported of a fish bone which had penetrated the esophageal wall and had become lost in the adjacent mediastinal structures.

The bone was successfully removed by peroral esophagoscopy.

Some of the dangers attending pointed objects which have perforated the esophageal wall are noted.

227 15TH STREET.

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LXV

THE PRODUCTION OF NEGATIVE PRESSURES BY RESPIRATORY CILIA

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In 1943 and 1944 Hilding[1] published the results of experimental work in which a negative pressure was developed in a ciliated mucous membrane lined cavity by the action of the cilia. This paper is a report of an attempt to corroborate that work.

J. Gray[2] in 1928 wrote an extensive monograph on the subject, "Ciliary Activity." In 1941 Proetz[3] devoted a considerable portion of his book, "Applied Physiology of the Nose," to a discussion of the activity of the cilia of the respiratory tract and of the nature and usefulness of the blanket of mucous which covers the cilia. More recently Ballenger[4] has reviewed the subject of ciliary activity.

As far as can be determined, Hilding is the only investigator to report on the ability of the respiratory cilia to create a negative pressure by their action under special circumstances.

Hilding's basic experiment involved an excised hen's trachea. Mucus first was collected from the tracheas of a number of hens and then was placed at the pulmonary end of the excised trachea in such a fashion that an occluding diaphragm of mucus was made.

The occluding diaphragm of mucus was moved up the trachea, presumably by ciliary action, and a negative pressure of 35-36 mm of water measured behind the mucus.

The experiment was repeated on a dog's frontal sinus by Hilding and Essex.[5] After anesthetizing the dogs with open drop ether, mucus was collected from the respiratory tract of the animal.

Through the roof of each frontal sinus a No. 18 needle was snugly inserted into the lumen of the sinus and connected to a water manometer. On the first side a second No. 18 needle was inserted into the lumen of the sinus and through this 3-7 cc of mucus was injected to the region of the orifice of the frontonasal duct. The second needle was closed.

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The position of the dog's head is not given in Hilding and Essex's paper, but judging from an illustration, the position is such that the frontonasal duct sloped slightly downward. Gravity, thus, would favor the development of a negative pressure.

In two of the three animals on which Hilding and Essex performed these sinus experiments, a negative pressure developed, the greatest being 66 mm H_2O. The negative pressure occurred after death or exsanguination.

Method: The present author, in the following experiments has attempted to develop a negative pressure in the excised tracheas of rabbits. Rabbits were killed with ether, the tracheas opened, and after a one-to-two-hour wait the mucus was collected from the pulmonary end. From four rabbits so prepared, approximately ½ cc of mucus was obtained.

An occluding diaphragm of mucus was placed in the pulmonary end of the excised trachea of a rabbit, which was then connected to a manometer.

Dogs anesthesized with ether were used in the second part of the experiment. The mucus was collected from the larynx by means of a long No. 18 needle inserted through the mouth. The animals then were allowed to die, due to an excess of ether or exsanguination. Additional mucus was then collected.

Trephine holes of .75 cm in diameter were made in the roof of each frontal sinus. Into one sinus 2-3 cc of mucus was injected about the orifice of the nasofrontal duct. Both holes then were tightly closed by rubber stoppers which carried No. 18 needles in their centers. Each needle was connected to a water manometer.

All of the animals were dead before commencing the experimental work, six by an overdose of ether and two by exsanguination. After placing the occluding mucus at the pulmonary end of the trachea and the manometer beyond that, negative pressures as high as 5 mm of water were recorded. Time and again the mucus diaphragm was seen to become increasingly thin and within 1-15 minutes to break so that the pressure returned to zero. The mucus apparently was removed by ciliary action from the periphery of the diaphragm. It would be transported to the laryngeal end where it could be collected and used again.

Reversing the position of the manometer resulted in positive pressures of approximately 5 mm of water.

If the manometer were not used, the intact diaphragm of mucus readily moved up the trachea to the opposite end.

TABLE 1.—THE NUMBERS REFER TO NEGATIVE PRESSURE AS MEASURED IN MM OF WATER.

Minutes / Animals	1	2	3	4	5	6	7	8	9	10	11
4	0	0	3	20	2.5	4.5	6	6	2	0	0
8	0	4.25	8	24	2	6	8.5	9	6	3	8
12	3	6.5	8	14	6	8	10	10	9	5	4
16	4	8.5	8	10	6	8.5	10.5	1	12	11	5
20	5	6.25		10	6	6	12		12.5	12.5	6.5
24	7.5	1		8.25	7.5	3	13		14	14	8
28	9			8.75	8	5	12		16	15	9.5
32	10.5			10	8				17	16	11.5
36	14			11					18	18	14
40	14			12					19	19.5	14
44	17			12					19	18.5	15
48	18			12					19	18	16
52	18			12					20	18	16.5
56	18			12					20	19	17
60				12					20		17
64				12					20		17.5
68				12					20		18
72				12							14.5

At the termination of all experiments the cilia could be seen on microscopic examination to be beating vigorously.

Hilding's experiments on the frontal sinus were repeated. There were 12 experiments performed on eight dogs. In two of the twelve experiments (No. 10 and 11), death by exsanguination occurred. In only one experiment (not included in table 1) did a negative pressure fail to develop.

Table 1 shows the results as they developed at four minute intervals. Figure 1 shows the average rise in negative pressure occurring after etherization (line B) and the two experiments in which death was due to exsanguination (line A). The negative pressure manifested itself in 10 of the 11 within 4 minutes. The greatest negative pressure developed was 24 mm and the average was 15 mm of water.

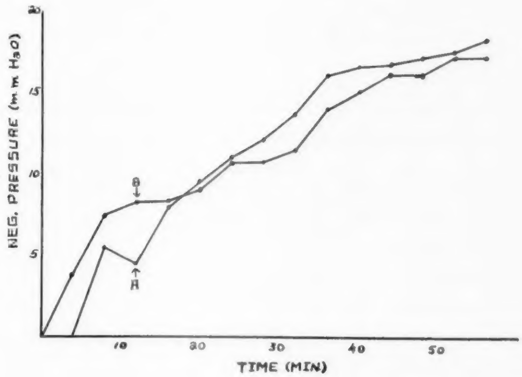

Fig. 1.

DISCUSSION

The degree of negative pressures developed in the present experiments was considerably less than those reported by Hilding and Essex. In the case of the trachea the difference was -5 mm H_2O compared to -35 to -36 and in the case of the frontal sinus the present experiments yielded an average of -15.5 mm H_2O, which is considerably less than the 66 mm H_2O, reported by Hilding as the largest negative pressure developed in his animals.

The explanation for the pressure differences is not readily apparent. The degree of negative pressure which can be supported by a diaphragm of mucus would be a function of the tensile strength of the diaphragm. The methods of collection of mucus for the experiments on the trachea were similar and so presumably the mucus should be of a similar nature. However, there is a possible source of error here.

There is no reason to suspect that there is a difference in the ability of the tracheal cilia of hens and rabbits to transport loads.

However, there are some factors which partly explain the pressure differences in the frontal sinus. Again the qualities (i.e. tensile strength) of the mucus bears a direct relationship to the pressure it will support.

The mucus in these experiments was collected from the larynx and the trachea. Hilding states that he collected it from the tracheas of dogs.

The nasofrontal duct in the present experiments was horizontal. In Hilding and Essex's work gravity probably favored the development of negative pressures.

In the method used by Hilding and Essex a smaller portion of ciliated mucous membrane was destroyed than by the method reported here. This would mean that by the latter method fewer cilia would be available to transport the mucus. However, in both methods the region about the orifice of the frontonasal duct and the duct itself was not disturbed.

CONCLUSION

By moving a mass of mucus the respiratory cilia can, under special circumstances, develop a negative pressure. The pressures obtained in these experiments were less than those reported previously.

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Clinical Notes

LXVI

NASAL SEPTAL HEMANGIOMAS

REPORT OF NINE CASES

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The recent removal of an ordinary nasal polyp during a routine polypectomy in the Out-Patient Department, which, on pathologic examination proved to be a hemangioma of the septum, prompted a review of the cases seen at the Massachusetts Eye and Ear Infirmary since 1928 when our first case appeared in the files. From that date to the present, over twenty-two years, only nine cases have been studied and are here reported. Ash and Old[1] in a recent paper described only fourteen cases from a group of twenty-three seen in over three thousand nasal polyp cases studied at the Army Medical Museum in the Registry of Otolaryngic Pathology.

CASE REPORTS

Case 1.—R. D., a thirteen year old boy, was first seen at the Infirmary on January 16, 1928 because of a nosebleed. Two months prior to admission he had his first epistaxis from the left nostril. He was treated by his local physician who cauterized an area on the left side of the nasal septum. Admission examination revealed a small, square centimeter sized, raised, red and crusted area on the lower border of the anterior part of the nasal septum at the level of the premaxilla. It was attached to the septum by a small base and had the appearance of granulation tissue. Removal was accomplished by sharp dissection and electrocautery. The Wasserman was negative. The pathology report was: Hemangioma.

Case 2.—Mr. J. J., age 81, was admitted to the Infirmary on August 17, 1934. For the previous three or four months he had noted increasing nasal obstruction and a slight amount of watery sanguineous discharge from the right nostril. Examination revealed a fleshy mass the size of a walnut attached to the right side of the septum. The exact location was not noted. Removal consisted of the routine submucous incision before and after the mass and use of the wire snare and electrocautery. The mass was removed intact with a "quarter-sized" area of septal cartilage exposed. The pathology diagnosis was: Hemangioma.

From the Department of Otolaryngology, Massachusetts Eye and Ear Infirmary, Boston, Massachusetts.

Case 3.—Miss M. J., age 40, was seen on December 10, 1934. Six months prior to admission she blew her nose and had a severe epistaxis. Four months before a mass was noted in the right vestibule. On September 25, 1934 a small polyp detached itself without bleeding. Three weeks prior to admission on forceful blowing of the nose considerable bleeding ensued for about half an hour with loss of about a cupful of blood. Examination revealed a fair sized mass somewhat pedunculated extending from the vestibule apparently attached to the septum. The mass was removed with a wire snare under cocaine anesthesia. The pathology diagnosis was: Hemangioma.

Case 4.—Mrs. H. K., age 58 gave a history of repeated epistaxis. On November 21, 1937, by use of diathermy, a hemangioma was removed from the left Kiesselbach's area.

Case 5.—Miss S. J., aged 44, was first seen on August 19, 1939. Two months prior to admission she began working with furs and had considerable irritation of the nose. On July 1, 1939 she noted a "pimple" inside the nose on the right. At first her local physician called it a "varicose vein," but after treatment it developed into an ulcer. The irritation persisted and a week later her physician reported that a polyp had developed at the site of the former "pimple." Bleeding had ensued and the right side was packed and she was referred to the Eye and Ear Infirmary for treatment.

Her general physical examination was negative. The nasal septum was deviated slightly to the right. Local examination revealed that the mass bled easily but seemed more fibrous in nature and not polypoid. Biopsy at this time revealed hemangioma. A week later a submucous resection of the septum was performed with excision of the mucous membrane over the anterior one-fourth of the septum. The operative site healed well and she was discharged well on the twelfth hosiptal day.

Case 6.—Miss R. R., age 24 years, gave a history of frequent slight nasal hemorrhages from the left side since tonsillectomy in July of 1949. Examination in the clinic revealed a pedunculated mass the size of a pea arising from the junction of the septum and the floor of the nose about an inch behind the vestibule. There was no history of injury or chronic irritation of the nose. The mass was removed under local cocaine and novocaine anesthesia and healing was uneventful. Pathology diagnosis was: Hemangioma.

Case 7.—Mr. D. S., age 69, had a mass in the left side of the nose removed eight months prior to admission on March 6, 1949. On this date it was found to have regrown posterior to the original scar. Its present location was on the septum opposite the tip of the middle turbinate. Removal was accomplished with a nasal snare and pathology diagnosis was: Hemangioma.

Case 8.—Mr. M. G., age 20, entered on February 5, 1949 complaining of epistaxis and nasal obstruction of about one month's duration. In November, 1948 during a football game he received a blow on the nose. Since that time there have been frequent nosebleeds after minor trauma. For the past month he had noted a thin watery pinkish discharge from the right nostril, enough to wet two handkerchiefs daily. Examination revealed a pedunculated mass at the mucocutaneous junction on the septum. After snare removal the base was cauterized with a silver nitrate stick. Diagnosis was: Hemangioma.

Case 9.—Mr. R. C., age 29, was seen in the out-patient department on April 30, 1950 complaining of epistaxis "off and on" for the past year with increasing nasal obstruction on the right. Examination revealed a thumb-sized mass completely obstructing the right naris with a tendency to bleed when touched. After routine cocainization a wire snare was inserted and the polyp was removed intact. On looking for a base to cauterize it was not found in the middle meatus as expected but on a septal spur about two centimeters behind the vestibule in the posterior part of Little's area. An oxycel gauze layer was placed over the raw surface effectively controlling hemorrhage.

DISCUSSION

The most frequent complaint in this small series was epistaxis, usually of frequent occurrence prior to admission. Of the nine cases this sign was present in five; the next most common complaint was partial or complete nasal obstruction.

The tumor occurred in adult life in all but one, the first case. This follows the known characteristic of this type of tumor in that it usually occurs in adults. Five of our cases were male; four, female. Clinically these tumors were either granulomatous or polypoid; usually fleshy with a tendency to bleed when touched with a probe. They were insensitive. In only one case, No. 5, was gross ulceration present. When polypoid they usually had more or less of a definite peduncle and this in every instance had its origin from the nasal septum in an area limited by the mucocutaneous junction at the columella, the floor of the nose, junction between bony and cartilaginous portions of the septum and superior margin of the septum. It is most likely that these tumors arose from the highly vascular Little's area.

There is no predilection for either side; five occurred on the right, and four on the left. The fact that in three cases, Numbers

1, 7, 8 and 9, the tumor arose from the posterior part of Little's area suggests the possibility that some may have arisen from the mucous membrane over the tubercle of the septum which is known to be highly vascular.

The lesion is definitely not related to the spider telangiectasis often seen in the nasal mucosa. These latter are considered to be hamartomas; that is, a local hypertrophy of one or more elements normally constituting a tissue: in this case the mucosal blood vessels, more particularly the capillaries.

Their predominant occurrence in adults rules out a congenital lesion. The fact that it tends to be circumscribed, noninvasive, and composed of well organized blood vessels of various calibers makes this lesion most likely a true neoplastic growth, fortunately benign. Ash and Old[1] believe that it is similar to the granuloma pyogenicum of the skin of the fingers and toes. In only one case, No. 5, could irritation have played a part in its causation.

Réthi[2] described five cases of "septum polyp" that originated from Kiesselbach's area. He considered it not a frequent growth. Treatment was by galvanocautery after cocaine and adrenalin. Weidlein[3] described three septal hemangiomas treated by surgical excision with no recurrences. He notes that recurrences have appeared after the cold snare technique. In our nine cases surgical excision, electrocautery, and wire snare removal were used alone or in combination as the occasion required. To date there have been no recurrences.

SUMMARY

Nine cases of septal hemangioma seen during the past twenty-two years at the Massachusetts Eye and Ear Infirmary are reported and described. This particular type of tumor is an adult lesion, noninvasive, circumscribed and usually composed of well organized blood vessels of varying sizes and calibers. Treatment was by use of electrocautery, surgical excision, or wire snare either alone or in combination as the occasion demanded. A description of the microscopic appearance of the tumor in the latest case is presented.

243 Charles Street.

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The Scientific Papers of the American Broncho-Esophagological Association

LXVII

THE PRESIDENT'S ADDRESS

LeRoy A. Schall, M.D.
Boston, Mass.

Members of the American Broncho-Esophagological Association and Guests:

As President of your Association, I have the honor to bid you welcome to the thirty-first annual meeting and to extend to you an invitation to participate in the discussions of the scientific papers that will be presented for your consideration.

May I express to the Association my appreciation for the honor you have bestowed upon me. To be enrolled in the company of distinguished men who have served as your president is indeed a privilege and a great honor. The years of this Association and my years in the practice of medicine are the same. Before this Association, at its meeting in Montreal in 1926, I gave (by invitation) my first paper before a national society. The kindness of your members to my maiden effort will never be forgotten. I recently re-read that paper—on the direction of the esophagus in Potts Disease and scoliosis—and now regret I did not save it for this Presidential Address.

To our hard-working Secretary, we owe an excellent program; to the Vice-President, to the Editor, to the Treasurer, and to the members of the Council, I am indebted for their guidance and support.

In reviewing the Transactions of this Association, one can trace the growth of broncho-esophagology as a specialty. All honor is due to those pioneers who, by their untiring efforts, developed this science of exploring the unknown regions by light and by sight. It was but natural that these early efforts were directed to the solution of the foreign body problem. It was natural, too, that this effort was lead by otolaryngologists into whose domain these problems arose.

It has been my privilege to have lived through the expansion of this specialty from that of the foreign body to that of disease. I vividly recall the long, hard battle of convincing the internist of the value of bronchoscopy in obscure pulmonary disease. In the early days of our pulmonary clinic, every bronchoscopy was the result of tact and artful persuasion as to why it should be done. Today all this is changed—the foreign body problem has receded into the background. The medical problems comprise the vast majority of our interest and no longer must we sell our diagnostic skill. It is only natural that this widening of interest in bronchoesophagology should interest others than laryngologists.

There are pessimistic souls and frustrated individuals who bewail this intrusion into what they would have us believe is the exclusive field of the laryngologist. Who can resent an internist with sufficient training and skill who desires to complete his examination by his own endoscopic work? Who can censor the thoracic surgeon who wishes to see for himself the problem for which he is going to operate? Surely this specialty is big enough for all who are willing to devote the time and the effort to acquire the skill to perfect themselves in its arts.

It was only in 1928 that my thesis based on the bronchoscopic diagnosis of six cases of primary carcinoma of the bronchi was accepted for membership by a national society. Today the cytologic examination of pulmonary secretions aids our diagnosis of carcinoma and the improved bronchoscopes have been of great value. It seems but yesterday that we were debating the use and the dangers of iodized oil in lung mapping. Today bronchograms have been simplified and are an accepted diagnostic procedure.

Within the past few years the problems of the persistant right aortic arch has been solved, diagnostically and surgically. Who can predict the limits of progress? Only by teamwork with other branches of the medical sciences will progress be made. Bronchoesophagology is not at the crossroads, but is on the high road of scientific progress. Let us have faith in the future of our calling and by our industry demonstrate that faith. Let us dedicate ourselves to the building of a better mouse trap and stop worrying about the path to the door.

243 Charles Street.

LXVIII

LOCALIZED PULMONARY HODGKIN'S DISEASE; BRONCHOSCOPIC ASPECTS

CASE REPORT

Clyde A. Heatly, M.D.
Rochester, N. Y.

Involvement of the lung as part of the progressive lymphadenopathy characteristic of Hodgkin's disease is common and well recognized. According to Versé, pulmonary lesions are present in 40 per cent of all such cases. Jackson and Parker[1] found pulmonary disease in 41 per cent of their series of 59 cases. Sternberg, Moolten and Longcope have made similar observations. The true nature of the pulmonary lesions in many of these cases is seldom in doubt. Painless swelling of the superficial lymph nodes especially in the cervical region appears as the first sign of the disease in most collected statistics and the diagnosis can readily be established by the histologic examination of an excised node. Less familiar is the occurrence of Hodgkin's disease localized to the thoracic cavity with little or no accompanying superficial adenopathy. It is this relatively small group of cases, usually unrecognized, which is often mistaken for commoner forms of pulmonary tumors. Rubenfeld and Clark[2] in 1937 reported such a case in which the limitation of the clinical signs to the lungs for five years before the appearance of adenopathy suggested a pulmonary origin for the disease. This case was first considered to be malignant until the dramatic response to roentgen therapy altered the diagnosis. Predominant pulmonary involvement in Hodgkin's disease has been reported by Versé, Wachner, Falconer and others, but convincing proof of a pulmonary origin of the disease according to Rubenfeld and Clark exists in only three instances. Moolten[3] however expresses the opinion that in fully 10 per cent of the cases with pulmonary manifestations, the clinical and morphological evidence would seem to indicate an actual origin within the lung substance.

An understanding of the rich distribution of lymphoid tissue within the lung is important in this connection. Pulmonary lym-

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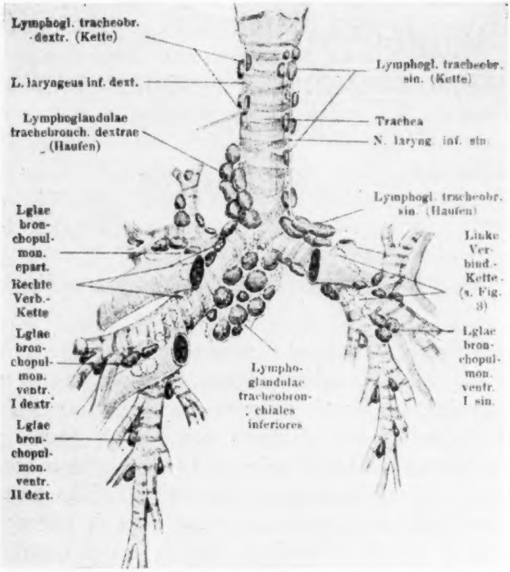
Lymphogl. tracheobr. dextr. (Kette)
L. laryngeus inf. dext.
Lymphoglandulae trachebronch. dextrae (Haufen)
Lglae bronchopulmon. epart.
Rechte Verb.-Kette
Lglae bronchopulmon. ventr. I dextr.
Lglae bronchopulmon. ventr. II dext.
Lymphogl. tracheobr. sin. (Kette)
Trachea
N. laryng. inf. sin.
Lymphogl. tracheobr. sin. (Haufen)
Linke Verbind.-Kette. (s. Fig. 3)
Lglae bronchopulmon. ventr. I sin.
Lymphoglandulae tracheobronchiales inferiores

Fig. 1.—Distribution of the tracheo-bronchial lymph nodes, according to Sukiennikow.

phoid tissue according to Miller[4] may be peribronchial, periarterial, perivenous or pleural and may occur in the form of nodes, follicles or small masses of lymphoid tissue. Lymph nodes are particularly abundant along the course of the larger bronchi and are quite constant at the points of bronchial branching (Fig. 1). In bronchi which have cartilages in their walls the lymphoid tissue may be situated between the cartilage and the tunica muscularis or outside of the cartilage in the peribronchial connective tissue. Similar collections are also to be found along the course of the pulmonary vessels particularly at their points of branching. The pleura contains a considerable amount of lymphoid tissue but according to Miller,[4] in the normal pleura, lymph follicles and nodes are absent.

The pulmonary lesions of Hodgkin's disease may be classified as mediastinal, parenchymal and pleural. Wolpaw, Higley and Hauser[5] in their series of 55 proved cases reported 63 per cent showing intrathoracic involvement and of these 50 per cent were mediastinal and 40 per cent parenchymal in distribution. The mediastinum contains an extensive collection of lymph nodes and limited or generalized involvement may occur. The hilar form is especially common.

Vieta and Craver in their study of 335 cases conclude that there are no specific roentgenologic features in pulmonary Hodgkin's disease. This is particularly true in the parenchymal types which present a wide variety of roentgenologic manifestations and may closely simulate tuberculosis, pneumonia, bronchogenic carcinoma, sarcoidosis, pulmonary abscess and pulmonary metastasis. Massive parenchymal lesions may result from direct infiltration by mediastinal nodes. A frequent mode of extension is by infiltration of the disease along the peribronchial lymphatics producing a granulomatous bronchitis and peribronchitis and presenting roentgenographic evidence of linear or feathery infiltrations radiating from the hilum. Lobar as well as lobular areas of consolidation may occur and cavitation is occasionally seen as the result of necrosis of granulomatous masses. A less frequent manifestation is the occurrence of well circumscribed isolated nodules which closely resemble the roentgen appearance of pulmonary metastases. Goldman[6] found 19 such instances in his review of 212 cases.

According to Moolten[3] who has presented one of the best descriptions of the bronchial aspects of Hodgkin's disease, a significant degree of bronchial involvement must be considered among the most typical features of its pulmonary lesions. In addition to the peribronchial extensions, granulomatous infiltration of the bronchial mucosa is common and may vary from a mild thickening grossly resembling an ordinary bronchitis to bulky, nodular outgrowths which narrow or completely fill the lumen. Vieta and Craver reported 5 cases in their series showing definite ulcerations or plaques of granulomatous tissue in the larger bronchi. In one of these cases a positive biopsy obtained at bronchoscopy led to the diagnosis of Hodgkin's disease. Polypoid growths within the large bronchi are rare and the case reported by Moolten[3] is in his experience unique. Compression or actual invasion of the bronchial wall from adjacent diseased lymph nodes may occur resulting in progressive atelectasis. Hurd[7] presented to this Association in 1922 an early case of Hodgkin's disease in which the evidence of bronchial compression obtained at bronchoscopy led to the establishment of the true diagnosis. The close similarity of many of these lesions to bronchogenic carcinoma must be stressed.

The clinical recognition of Hodgkin's disease becomes extremely difficult when the lesions are restricted to the thoracic cavity. The following case is illustrative.

C. H., an eighteen year old male, was admitted to the Strong Memorial Hospital on March 26, 1949 complaining of cough and shortness of breath. Two years before he had had a roentgenogram

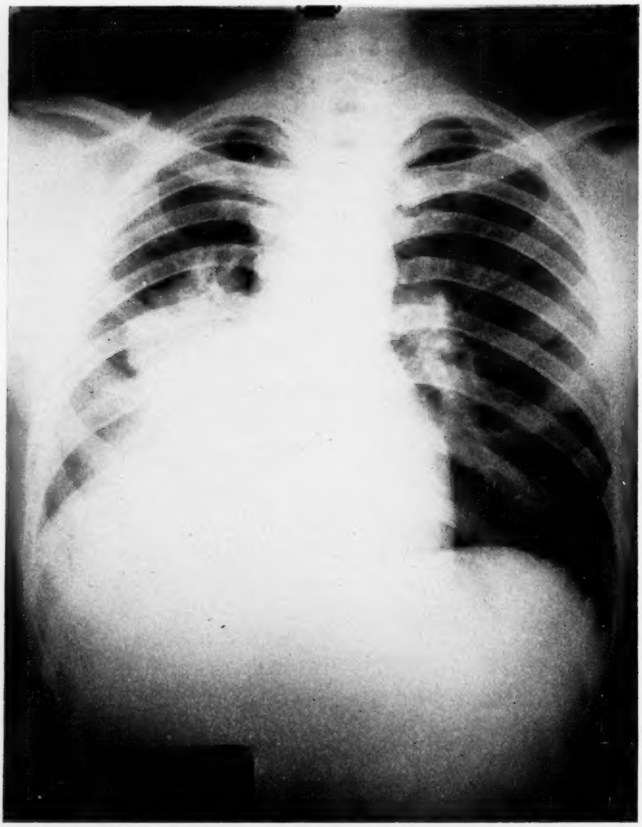

Fig. 2.—Anteroposterior view showing hilar mass invading the right mid lung field with atelectasis of the lower lobe.

of his chest taken by a mobile unit in Vermont. A repeat film was taken and a third requested but never made. The patient did not know the result of these examinations. The present illness began one week before admission with sudden onset of cough and dyspnea on exertion. There was no associated pain nor hemoptysis. Clinical examination showed evidence of atelectasis of the right lower lobe with shifting of the mediastinal contents to the right side. There was mild fever (38.2C) and leukocytosis (11,000). The differential blood count was normal. Roentgen examination confirmed the atelectasis of the right lower lobe and also showed a large circumscribed area of increased density in the right mid lung field which was considered to be due to a neoplasm (Fig. 2 and 3). The spleen was not enlarged and a careful search disclosed no evidence of lymphadenopathy. Three days later a bronchoscopic examination was performed. Some widening of the carina was noted. Just distal to the orifice of the middle lobe branch, the right main bronchus was found to be completely occluded by a smooth, slightly reddish mass which grossly appeared not unlike a bronchial adenoma. A biopsy was followed by rather free bleeding and troublesome coughing. Microscopic study of this tissue showed a fibrous stroma in which there were numerous polymorphonuclear and mononuclear cells as well as scattered larger cells with round or oval granular nuclei and a rather pale blue staining cytoplasm. The character of these larger cells was considered suggestive of some form of lymphoma, possibly Hodgkin's, but a definite diagnosis could not be made. Cultures obtained from the right bronchus showed no evidence of fungus or tubercle bacilli. In an effort to establish a diagnosis, a second and third biopsy were obtained bronchoscopically with identical inconclusive results. A careful search was made for superficial glands for section purposes but none were palpable. Meanwhile, although the pneumonitis in the atelectatic lung was being controlled by antibiotics, the nodular shadow in the mid lung field seemed to be increasing slightly. Exploration of the right chest with the view of possible pneumonectomy was therefore decided upon. On May 2, the right chest was opened. The pleural cavity contained a moderate amount of straw colored fluid. The right lower lobe was completely atelectatic. In the right hilar areta a solid tumor mass was discovered about the size of a baseball extending into the middle lobe and densely adherent to the pericardium and inferior vena cava. Several attempts were made from different angles to effect pneumonectomy but the dense adhesions prevented. The operation was discontinued after securing a biopsy from the tumor mass. Histologic examination of this tissue showed typical Hodgkin's disease with characteristic eosinophilic infiltration and Dorothy Reed giant cells. The postoperative course was uneventful. Radiation therapy was begun with the immediate temporary improvement usual to these cases.

Conclusions: Pulmonary involvement is frequent in Hodgkin's disease and in typical cases the associated enlargement of superficial lymph nodes permits accurate diagnosis by gland biopsy. Uncertainty however may arise in cases in which external lymphomas are poorly developed or altogether absent. It is important to emphasize that the lesions of Hodgkin's disease both clinically and roentgenologically may closely simulate many other pulmonary conditions. A significant degree of bronchial involvement must be considered

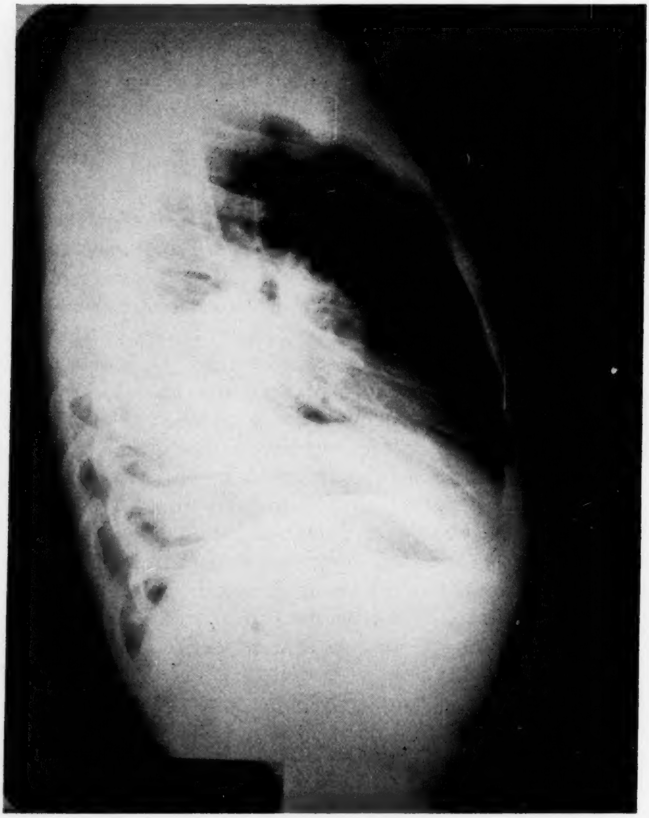

Fig. 3.—Lateral view of preceding.

among the characteristic features of the pulmonary manifestations of the disease. This may take the form of external compression of the bronchus or infiltration and invasion producing endobronchial lesions which may closely resemble carcinoma or adenoma. Occasionally, as in the case reported, even the histologic studies of biopsied material may not be entirely conclusive. The response of Hodgkin's granulomata to roentgen therapy may furnish valuable evidence in the differential diagnosis of these more obscure cases.

11 North Goodman Street.

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DISCUSSION

Dr. Louis H. Clerf (Philadelphia, Pa.): I recall several interesting patients that had definite bronchologic findings and in two it was possible to make a diagnosis of granuloma on the basis of biopsy. The pathologist was not able to make a positive diagnosis of Hodgkin's disease, although in one case he suspected it. The bronchopulmonary findings were the first clinical manifestations. There were no demonstrable lymphnodes although later both patients did develop lymphnodes in the neck. Both patients were young men under 20 years.

In a third case, a girl aged 16 years, had a supraclavicular node and clinical evidence of bronchial obstruction. The pathologist made a diagnosis of Hodgkin's disease on the basis of a bronchoscopic biopsy.

We must realize that Hodgkin's disease is not entirely a disease of lymphnodes in the neck. Mediastinal nodes share equally with the cervical nodes. We often find bronchial and tracheal compression so the bronchologist often will be in a position to sugest to the internist, as he is the one most often involved, that there is a possibility of Hodgkin's disease. I would like to emphasize at this time the importance of the bronchologist in the field of general chest diagnosis and his ability to help the internist interpret obscure clinical findings. If one simply describes what is seen at the end of the bronchoscope it may mean little to the internist and thoracic surgeon. We should have a comprehensive picture as to just what the problems are, and if we can do that, the internist and thoracic surgeon will have less need for a bronchologist on their own personal staff.

LXIX

REMARKS ON THE INDICATIONS FOR BRONCHOSCOPY AND ESOPHAGOSCOPY FROM THE STANDPOINT OF THE INTERNIST

ARTHUR L. BLOOMFIELD, M.D.
SAN FRANCISCO, CALIF.

(Abstract)

I want to say first of all how much I appreciate the invitation to speak before the American Broncho-Esophageal Association. Needless to say, it would be out of order for me to attempt discussion of any technical aspects of the indications for endoscopy before a group of experts of this sort. There are, however, a few points about the use of diagnostic procedures in general which I consider worthy of mention.

The recent rapid development of new diagnostic tests and technical procedures has created for the physician a real embarrassment of riches. Furthermore there are strong pressures to make the laboratory and technical study of the case more and more complicated. Not only does the doctor wish to be sure that he is up-to-date and doing everything thoroughly, but the enthusiasm of drug houses and instrument makers is almost impossible to resist. After a certain amount of literature has been received telling how easy it is to diagnose a certain disorder by means of a new drug the natural tendency is to follow this suggestion; if a pocket electrophoresis apparatus should appear on the market it would not be hard to persuade the doctor of the usefulness of knowing the protein patterns in various diseases. Now most of these tests are useful and progressive; the bad thing is indiscriminate and inappropriate use of them. At the moment, for example, there is a widespread tendency among house officers, after having recorded the history and physical examination, to enumerate under the heading of "suggest doing" an extravagant list of tests and procedures of every sort, often without very definite indications for their use. The idea seems to be to anticipate any suggestion for further study which may possibly come up. That this tendency, which we choose to speak of as "peripheral medicine" in distinction to the essential central matters of careful history and physical examination followed by judiciously selected tests which are chosen as one goes along because there is reason to believe they may actually answer a question posed by the case, that this tendency is unsound was anticipated even many years ago by a great clinician upon whose comments we cannot improve:

> "To give a medical opinion is not an academic exercise. Utility is implicit in it—no method is so simple as to be worth employing if it can yield no valuable result for the patient, and none is too elaborate if it can save life or in the slightest degree shorten illness or point the way toward health. It is not scientific to employ every possible method of investigation in the examination of every patient, but is meddlesome, is usually dishonest—and where thus employed clouds, not clarifies, the judgment."*

Not only the house officer, but the visiting man is likely to succumb to the temptation of the laboratory "bill of fare" offered by his hospital and to have a good many tests performed simply because they are readily available. "Oh yes, let's get a blood iodine, and we may as well get a sodium and potassium and check the potassium with an EKG, and we may as well get an acid phosphatase and skull films and an EEG, etc. etc." is the sort of conversation heard daily in any hospital corridor between chief and house officer.

You have asked me to speak to you on the indications for bronchoscopy and esophagoscopy from the standpoint of the internist and the tone of my remarks is, I am sure, already clear to you from what we have said above. The point of major importance in the use of these procedures is not only their intrinsic value but their use at the proper time in the course of studying the case. This makes for that elegance and artistry which is inherent in the best clinical practice. Tables 1 to 4 summarize briefly a few of the situations in which bronchoscopy and esophagoscopy may be useful to the general doctor. We believe the endoscopist should not be asked to operate purely as a technician; he should always be regarded as a consultant and should pass on the problem himself before making the examination. There has grown up some confusion as to just who should perform bronchoscopy in cases of pulmonary disease. The professional endoscopist is presumably more dextrous; the chest physician on the other hand has special knowledge of the pathological problem. There is, of course, no absolute answer and here again the internist, as the entrepreneur, must "call the signals" and use his judgment as to which availaible endoscopist will best meet the needs of the case.

STANDFORD HOSPITAL.

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*Janeway, T. C.: Canad. Med. Ass'n Jour. 7:589, 1917.

TABLE 1.

INDICATIONS FOR LABORATORY TESTS

1. Certain tests should be done in every case—
    - Blood count
    - Urine examination
    - Stool examination
    - Wassermann?
2. From this point on tests or procedures should only be done if they may answer a question posed by the case. Beware of peripheral medicine. Bronchoscopy and esophagoscopy come under this head if not appropriately used. The point is not only their intrinsic value but to use them at the proper time in studying case.

TABLE 2.

BRONCHOSCOPY — INDICATIONS

Diagnostic.

1) To diagnose presence of disease or its nature—
    - a) by appearance
        - Ex. Tumor
        - Bleeding
        - Foreign body
    - b) by biopsy
        - Ex. Tumor
        - Inflammation

2) Disease known—to define status.
    - Ex. Condition of bronchi in tuberculosis.
    - Patency of abscess cavity, etc.

3) To localize disease.

Prognostic.

To follow course of case.

Therapeutic.

1) Removal of foreign body.

2) Aspiration of secretions.

3) Introduction of medicaments.

TABLE 2.

ESOPHAGOSCOPY

1. *Diagnostic.*
    - a) By appearance.
        Ex.—Carcinoma
        Spasm
        Varices
        Diverticula
    - b) By biopsy.
        Ex.—Carcinoma
        Inflammation
2. *Prognostic.*
    Follow course of case.
    Ex.—Cardiospasm
    Ulceration
3. *Therapeutic.*
    - a) Removal of foreign body.
    - b) Injection of veins.

TABLE 4.

Objections to Bronchoscopy and Esophagoscopy from the standpoint of the internist.

All the complexities of arranging an operation.

An ordeal for patients in poor condition or in old age. Fear.

May be dangerous in some cases—aneurism, etc.

Postoperative discomfort.

Expense.

Advantages.

An invaluable emergency measure—foreign bodies.

An inevitable diagnostic step at the proper point should usually follow history, physical examination, sputum examination, x-rays and cytology.

DISCUSSION

Dr. Frederick T. Hill (Waterville, Maine): It seems to me that this presentation of Dr. Bloomfield's shows fine common sense and a logical presentation of something that is very important. He calls for the pooling of professional skills in handling these diagnostic problems. First things come first. I am particularly impressed with his admonition to beware of "peripheral medicine," in the hospital, with too much emphasis upon the laboratory. Also when we remember that sometimes "peripheral medicine" is encountered when obvious procedures are neglected. It brings to my mind an experince we had a short time ago where a patient who had been unable to swallow for five days was sent into the hospital. He had become dehydrated and during this time had been treated on the outside with penicillin. He had a large impacted esophageal foreign body. No attempt had been made to ascertain the cause of his obstruction.

LXX

MAGNET EXTRACTION OF FOREIGN BODIES

EDWARD J. WHALEN, M.D.
HARTFORD, CONN.

The physical phenomenon of magnetic force was known to the ancients who discovered that a strange kind of ore found in the province of Magnesia in Greece would attract small bits of iron. It was called magnet stone because of its place of discovery. When a bar of iron was stroked with the magnet stone, the bar in turn took on the magnetic qualities of the stone. This phenomenon remained a curiosity until sailors visiting the China ports learned from the Chinese that one end of a magnetized particle of iron always turned to the north. Sailors soon made use of such a magnetized metal as a direction finder and termed it a lodestone or leading stone. Columbus understood and made use of such a magnetized needle as a compass on his many voyages. During this same era it had been found that by rubbing silk or amber in a vigorous manner, particles of paper or other light objects would exhibit attraction. Of all the phenomena of electricity and magnetism, frictional electricity and ferro-magnetism are the oldest known to man and the least understood today.

Such a mysterious force made a strong appeal to the ancient physician and frequent reference is made to the use of the magnet in early medical writings. Avicenna in the ninth century taught that iron taken internally was a poison and as an antidote he recommended the magnet in doses of one grain which by uniting with the iron of the body would render the poison inert. For the cure of hernia it was advised that iron filings be administered to the patient followed by the application of a magnet plaster in the belief that the attraction of the magnet in the paster for the iron would draw the protruding intestine and aid in its replacement. No less a surgeon than Ambroise Paré reported a hernia that was relieved by this method.

Such was the use made of magnetic force in medicine and surgery until late in the 18th century when Galvani found that the leg of a dead frog would twitch when brought in contact with certain metals, the original muscle nerve preparation. Franklin had

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Read before the Annual Meeting of the American Broncho-Esophagological Association, San Francisco, May 25, 1950.

recently shown that lightning was electricity and Volta was experimenting with a galvanic pile that was eventually to become the electric battery. This rich store of information about magnetism and electricity uncovered by true scientists was at once grasped by the quacks and charlatans. Mesmer provided his patients with magnets which they held in the hand while in a state of hypnosis only to come out of the hypnotic trance with the same psychosomatic complaints which they brought to the seance.

Elisha Perkins, a Connecticut Yankee doctor, devised a method of magnetism for drawing out of the body the disease elements with a pair of metallic, king-size toothpicks which he called Perkins' Tractors. These tractors were sold throughout the world and brought a fortune to the doctor. A pair was sold to George Washington for the sum of thirty dollars.

During the next century, the use of magnetism in medicine was limited to the use of the electromagnet for the removal of metallic foreign bodies from the eye.

The removal of foreign bodies by means of a magnet is not a new procedure but rather a neglected one. Dr. de Roaldes in 1900 described a technic for the removal of a magnetic foreign body from the trachea by means of a Haab magnet attached to a soft iron rod. Dr. Chevalier Jackson in 1905 published a comprehensive analysis of the method of magnet removal of foreign bodies form the air and food passages, an analysis that today might well be used as a guide for the technic of foreign body removal. The only addition that has been made is the use of the Alnico magnet which was perfected many years following the Jackson survey in 1905.

Magnets are divided into two types, the permanent magnet which retains for an indefinite period its magnetic force and the electromagnet by which a magnetic field can be created by the passage of an electric current and the field broken when the electric current is shut off. We think of the permanent magnet in terms of the horseshoe magnet, the toy of our younger days. The electromagnet is familiar to some of us as the magneto in the automobile of the first years of this century.

The fact that a strong ferromagnetic alloy could be formed from nonferromagnetic constituents was discovered by Hensler in 1898. In 1940 physicists perfected a new type of permanent magnet by the combination of iron, cobalt, nickel and aluminun, a magnet having a long life and coercive force much greater than was found in the old type permanent magnet. Under the trade name, Alnico, this magnet has found wide usefulness in industry and now is used in airplanes, loudspeakers, television sets and lastly in our endoscopic armamentarium. Because of the great coercive force of the Alnico magnet and its small size in relation to this great magnetic force, it has been found suitable for use in the limited spaces of the air and food passages.

In outlining the treatment of metallic foreign bodies of the air and food passages, it may be that the magnet removal of the object will be given consideration as well as the established method of forceps removal. Whatever method is planned for such removal, it is of prime importance that a duplicate of the foreign body be obtained. A study of the duplicate and a survey of the x-ray films of the organs involved will permit us to test a like object to determine if the foreign body be magnetizable.

The Berman Locator is helpful in determining if the metallic foreign body will be attracted by the pull of a magnet. In a case of a metallic foreign body of the air or food passages, the Berman Locator, if passed over the area adjacent to the foreign body, will respond with a signal if the object is magnetizable.

Foreign bodies of the air passages are for the most part approached by the more conventional method of forceps removal. A small number of cylindrical foreign bodies such as straight pins, snare wire and bobby pins are aspirated into the finer divisions of the bronchial tree beyond the point where they can be approached by forceps. It is in these cases that an Alnico magnet attached to the end of a rigid rod as planned by Holinger, or the vertebrated type described by Jackson, which permits entrance to the second division of the bronchi, can be brought in contact with the foreign body under fluroscopic guidance and be removed. A clear understanding of the segmental orifices and the relation of the bronchopulmonary segments helps to simplify the removal of foreign bodies from the finer divisions of the bronchi by means of the biplane fluroscope and the permanent magnet. A recent improvement in the biplane fluroscope making use of two tubes with one screen to give a stereopticon effect has made more effective the use of the fluoroscpoe in the approach to the problem of foreign bodies in the deeper recesses of the bronchial tree.

The successful use of the magnet for the removal of metallic foreign bodies from the gastrointestinal tract has confirmed the possibilities of this procedure. It has been the opinion of some endoscopists that the magnet removal of such objects was limited to those foreign bodies present in the stomach. Recent reports of the successful magnet removal of foreign bodies from the duodenum

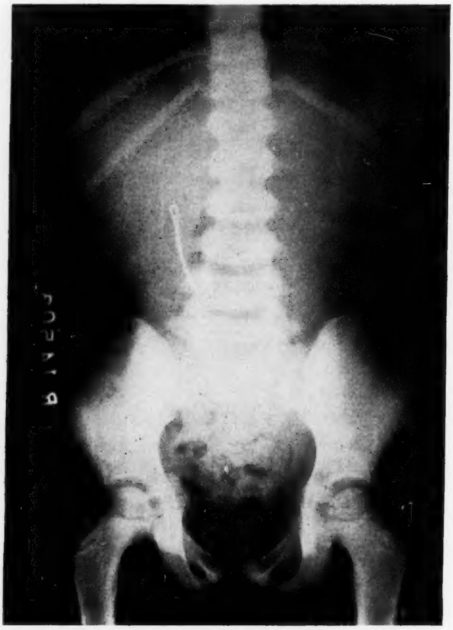

Fig. 1.—Foreign body, bobby pin, 5 days after ingestion. The fourth portion of the duodenum.

indicate that this procedure could be followed in the removal of magnetizable foreign bodies from the entire gastrointestinal tract.

Though it is an established principle that any object that can pass the cardia and enter the stomach will pass the pyloric sphincter, there are exceptions to this rule. Foreign bodies because of size, irregular shape or objects having sharp points, may remain in the stomach for a long period. The majority of gastric foreign bodies are metallic and in many of these are ferromagnetic. The gastroscopic removal of foreign bodies is possible and Tucker reports success in removing such objects with his magnet tipped gastroscopic forceps. Many endoscopists find this method to be technically difficult.

Foreign bodies of the stomach usually occur in children and are for the most part metallic. The usual routine is followed in these cases, namely, to obtain a duplicate of the foreign body and determine if the object is magnetizable. An x-ray study of the abdomen is then carried out using a carbonated beverage which gives a more satisfactory outline of the stomach than that obtained with barium or other opaque media.

Having determined that the foreign body is in the stomach and is magnetic, we now proceed to the insertion of the magnet into the stomach. Since this part of the procedure is done under fluoroscopic control, explosive anesthetics are not used. Under intravenous pentothal anesthesia, the esophagus is exposed with a speculum and the magnet, attached to a Levin tube is guided by forceps to the lower esophagus or stomach under fluroscopic guidance. A piano wire stylet in the tube permits control of the magnet. Under this control, the magnet will, in a few minutes, make contact with the foreign body and can be withdrawn. Under pentothal anesthesia, the foreign body is not stripped off at the cardia or cricopharyngeus. Since the entire procedure is under fluroscopic control, we are prepared to recover with forceps the foreign body if it loses contact with the magnet during the removal. This method of removing ferromagnetic foreign bodies from the stomach presents no technical difficulties and it works.

Many foreign bodies entering the stomach remain in this organ for only a few hours and then will be found to have passed the pylorus. In infants and children such a foreign body may have difficulty in passing through the duodenal section of the bowel. The first limb of the duodenum runs upward and backward, the second limb runs downward and the third limb passes from right to left and upward. It is due to these turnings and angles that a foreign body frequently becomes impacted and fixed in the duodenum. An additional barrier in this area is the ligament of Trietz. This structure, the suspensory ligament of the duodenum, acts to prevent the duodeno-jejunal flexure from being dragged down by the weight of the jejenum and limits the movement to make this a fixed point and an additional cause for a foreign body being impacted in the duodenum.

A foreign body trapped in the turns of the duodenum presents a major surgical problem since the surgeon finds it difficult after the abdomen has been opened to locate the foreign body in the thick walled retroperitoneal duodenum. It is technically difficult to incise this art of the duodenum and even more of a surgical problem to suture the duodenal incision at this point. For these reasons, the surgeon will sometimes seek the aid of the endoscopist in the treatment of such cases. Fortunately the endoscopis is in a position to cite many cases of successful removal of ferromagnetic foreign bodies from the duodenum.

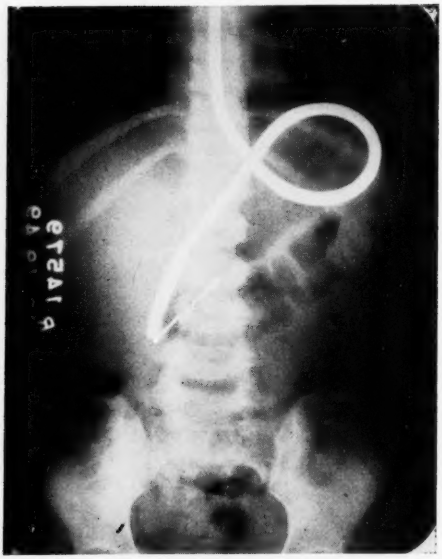

Fig. 2.—Foreign body, bobby pin, Alnico magnet attached to Cantor tube in contact with bobby pin, 15 hours after insertion of magnet.

The present routine use of duodenal tubes of the Levin or Cantor type and the Miller-Abbott tube used for intestinal decompression has shown us the feasibility of intubing the entire gastrointestinal tract as well as indicating the rate of speed with which the tube advances and the usual points of obstruction. This information is essential in the magnet removal of foreign bodies from the duodenum and other parts of the bowel.

The procedure followed in the magnet removal of duodenal foreign bodies is similar to that used in gastric foreign bodies. Having identified the location of the object, and being certain of its magnetizability, the Alnico magnet attached to a Cantor tube is placed in the stomach with forceps under fluoroscopic control, while the patient is under pentothal anesthesia. The magnet will have made its exit from the stomach and passed on to the duodenum in from 4 to 6 hours and will then advance rapidly through the duodenum. Because of the angulations of the duodenum, the Alnico magnet which is 3.5 cm in length may be obstructed at the same point as the foreign body but will have made contact. When contact has been confirmed by fluroscopic examination, the tube, magnet and foreign body are withdrawn.

The use of a permanent magnet of the Alnico type for the removal of metallic foreign bodies has one drawback, a major one. A foreign body attached to a permanent magnet cannot be detached nor can its presenting part be altered. This objection has been overcome by the use of an electromagnet and for this, Penta has devised an ingenious method of attaching a specially wound electromagnet to a plastic tube and connected to a six volt series of batteries. By use of this device, the magnetic foreign body in the stomach in contact with the electromagnet can be moved, after contact, to various parts of the stomach until a relationship of magnet to foreign body favorable to removal is achieved.

This is the story of Sandra, a six year old child who swallowed a bobby pin she had been holding in her mouth. The child reported the incident to her mother Wednesday. An x-ray study was carried out the following day, Thursday, at which time, it was reported that a foreign body, the shape of a bobby pin, was seen on the x-ray film and was described as being in the second portion of the duodenum. The child was admitted to the hospital for observation. No complaints were expressed by the patient and nothing unusual in her condition was observed by those in attendance. Friday, another x-ray examination showed the bobby pin to be in the same position, the second portion of the duodenum. On the fourth day, x-ray examination showed the pin to be in what was thought to be the fourth portion of the duodenum.

Sunday, the fifth day following the ingestion of the pin, a Cantor tube was passed through the nose and removed by way of the mouth so that an Alnico magnet 3.5 cms. in length could be attached to the tube. The child was under penthothal anesthesia during this and the successive steps of the procedure. The esophagus was exposed with a speculum and under fluroscopic guidance the Cantor tube with magnet attached was carried by forceps to the stomach. Following the arrival of the magnet and tube at the pylorus, 12 cms more of the Cantor tube was allowed to curl in the stomach to permit the egress of the tube to a point in the duodenum where the bobby pin had come to rest. The child was returned to bed and experienced a restless night with some vomiting of bile stained material. Monday morning, six days after the pin had been swallowed, a fluroscopic examination indicated that the magnet attached to the Cantor tube had made contact with the bobby pin. Without anesthesia and with frequent fluroscopic observations, the Cantor tube was withdrawn to the hypopharynx. The next step was to expose the pharynx and with a hemostat remove the tube, magnet and bobby pin. The magnet was then removed from the Cantor tube which allowed the tube to be withdrawn by way of the nose. The child was discharged from the hospital the following day after having been on a regular diet the previous twenty-four hours.

A discussion of the subject of magnet removal of foreign bodies would be incomplete if reference was not made to the work in this field by Dr. Murdock Equen. Doctor Equen has repeatedly called the attention of endoscopists to this simple and effective technic of dealing with magnetizable foreign bodies. His writings on this subject have been an important factor in establishing this as an accepted method for the solution of what is sometimes a baffling problem. The magnet removal of foreign bodies of the terminal bronchi, the stomach and other parts of the gastrointestinal tract remains an accomplishment still valued for its rarity.

The armamentarium of an endoscopist should include an Alnico magnet that can be attached to a duodenal or intestinal tube. A vertebrated tip magnet for use in the smaller divisions of the bronchial tree will, on occasions, be the best method for the removal of magnetizable foreign bodies from the bronchi. A Penta type electromagnet for the removal of ferromagnetic foreign bodies from the stomach will complete the endoscopic magnet equipment.

With the improvement in the Alnico permanent magnet and the simplification of biplane fluoroscopy together with our increased knowledge of intestinal intubation and the bronchopulmonary segments, endoscopists are now in a better position to make use of the simple and effective technic of the magnet removal of ferromagnetic foreign bodies from the air and food passages.

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DISCUSSION

Dr. Murdock Equen (Atlanta, Ga.): I think Dr. Whalen's paper is an excellent one; it should make bronchoscopists and general surgeons more conscious of the magnet.

You would be surprised how many inquiries we get about the magnet, "Where can I get the magnet?" We have sent many of them to bronchoscopists by plane but when the magnet gets there, so often a surgeon has beat the endoscopist to it and the stomach has been opened and the foreign body removed. If we can hold these surgeons down long enough until the magnet has been introduced, many a child will be saved a gastric opening.

The simplicity of the Alnico magnet makes it to me still the instrument of choice. Its use entails no cumbersome batteries, no connecting wires and no switches.

When a child swallows an open safety pin, the pin must go down, ring first, point trailing. If the pin is still in the esophagus, the alloy magnet on a fairly stiff wire can lead it down into the stomach, the point up. To get the pin out it is necessary to reverse it so that the ring is up and the point trailing. To do this you can pull back the magnet to strip off the pin at the cardia—for the power of the magnet is not great enough to cause the point to perforate the stomach—and then push the magnet back; or you can manipulate or flip the magnet in the inflated stomach, under fluoroscopic guidance, so that it engages the pin again, ring up, point down.

To remove a magnetic foreign body from the lung we use a small magnet, 3 mm in diameter, fixed in a ureteral catheter. Such a magnet may be passed into the smaller bronchioles; it requires less manipulation and therefore causes much less trauma than forceps.

We have not found it necessary to use our biplane fluoroscope for some years. We also think that ether is a more satisfactory anesthetic for bronchoscopy than pentothal or local agents because of the good relaxation.

We have never had trouble with sparking from the x-ray machine while the magnet is being used under fluoroscopic guidance.

Dr. Gabriel Tucker (Philadelphia, Pa.): I enjoyed Dr. Whalen's presentation very much. The flexible forcep, with the addition of the Equen magnet, has been found useful in the removal of magnetic foreign bodies from the stomach. This instrument could be properly called, the Equen-Tucker flexible magnetic forcep. Dr. Equen's curved magnet has been attached to the sheath of the flexible forcep in such a position that the non-magnetic forceps can be passed along side the magnet holding the foreign body in position. This is done under fluoroscopic guidance. In addition to the attraction of the magnet, the grasp of the forceps allows the foreign body to be brought up through the esophagus without trauma. The curved magnet attached to the sheath of the flexible forceps is introduced under fluoroscopic guidance into the stomach. When the stomach is inflated, and the patient placed in the proper position, this instrument can be passed into either the pyloric antrum or the stomach fundus. In a recent case, an open non-magnetic safety pin entering the pylorus, point first, was turned so that the spring passed into the duodenum with the point trailing. This pin passed through the gastro-intestinal tract without complication. By the manipulation, the advancing point was converted into a trailing point and possible perforation of the bowel, perhaps necessitating laparotomy was avoided.

LXXI

RUPTURE OF THE TRACHEA DURING BRONCHOSCOPY AND PRINCIPLES OF POSITIONING FOR ENDOSCOPIC PROCEDURES

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AND

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Rupture of the trachea during bronchoscopy is an extremely rare complication. The literature contains no report of it. Factors contributing to the rupture in the case we present, were the uncontrollable movements of the child which resulted in vertical movement of the head without rotation. The important factors influencing the successful treatment, were oxygen therapy, bilateral chest aspiration and mediastinotomy. The following is the detailed case report:

L. P. was a 7½ year old white male, weighing approximately 70 pounds. Indications for the bronchoscopy comprised a chronic productive cough and hoarseness of four months duration which followed an upper respiratory infection, a positive tuberculin patch test (at 1 to 1,000 dilution), and x-rays of the chest which revealed increased lung markings, bilaterally, with patchy infiltrations suggesting a bronchitis or a low grade pneumonitis. The boy was physically active and of the hyperkinetic type. The cervical lymph nodes were enlarged and the nasal mucosa was boggy and pale. Gastric washings did not reveal tubercle bacilli.

Premedication for the procedure consisted of 1/12 grain of morphine sulphate by hypodermic and 1½ grains of nembutal by mouth. No general anesthesia was employed. The child seemed extremely tense and apprehensive. The larynx was exposed with the child-size standard Jackson laryngoscope. Hyperemia of the mucosa was noted, with some thickening of the vocal cords. The 5x30 Jackson-Holinger bronchoscope with the extra small size lamp was introduced, during which time the child became uncooperative. In spite of the shoulder holder, the patient moved his shoulders

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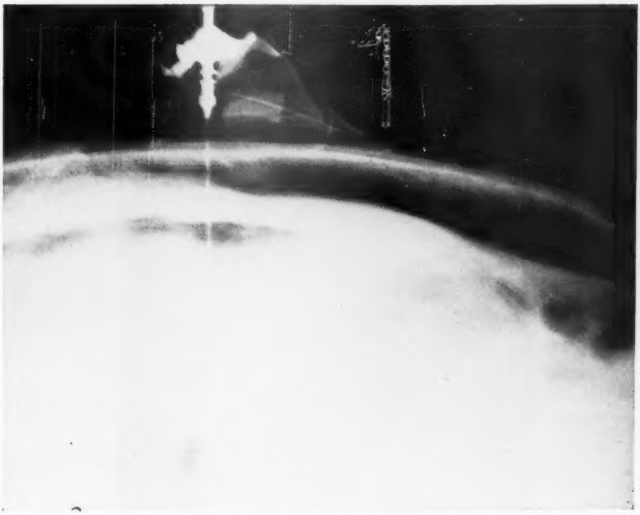


Fig. 1.—Right lateral recumbent chest x-ray (portable) after insertion of needle into right pleural space. The massive subcutaneous emphysema and the position of the needle in the right chest are demonstrated.

rather vigorously up and down. After removal of the laryngoscope, leaving the bronchoscope in the trachea, there appeared to be a web across the right posterolateral area of the field. A small amount of blood was seen, therefore, the procedure was immediately terminated. After the withdrawal of the scope, the child appeared to be in good condition and was returned to his bed.

Within three minutes, the face and neck were swollen, the swelling rapidly progressing to involve the entire body, head, and extremities. This subcutaneous emphysema when correlated with the bronchoscopic findings of a tear, suggested the diagnosis of rupture of the trachea. The child was extremely apprehensive and was unable to talk, although the voice was clear. He was restless and restraint was initially necessary. He seemed to be conscious through the entire episode and during the period of active treatment. Inspection of the mouth and throat revealed no abnormalities. The respirations were labored and the child became slightly cyanotic. The heart rate increased and the pulse was of fair quality. The swelling about the neck and chest was doughy in texture and crepitation was present only at the advancing margin of the subcutaneous emphy-

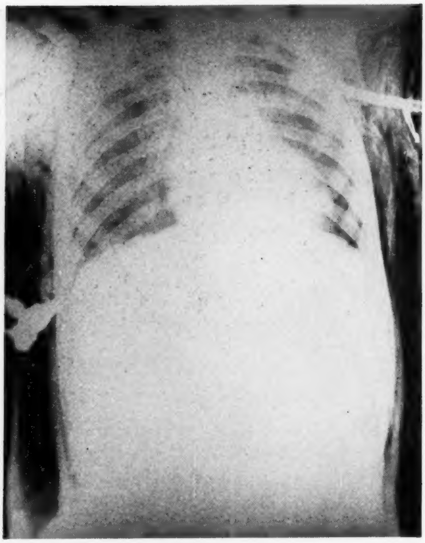


Fig. 2.—Antero-posterior chest x-ray (portable) after insertion of the catheter. The massive subcutaneous emphysema, the positions of the needle and catheter, and the superior mediastinal emphysema are shown. The lungs have re-expanded.

sema. Auscultation at the open mouth did not reveal stridor or wheeze. Blood pressure determinations were not made because of the swelling of the extremities. "100%" oxygen administration was started immediately using the oronasal BLB mask.

The patient was returned to the operating room and a long needle was inserted into the right pleural space. Aspiration demonstrated the presence of a pneumothorax and underwater drainage was established. Air continued to bubble out under water. Because the extreme swelling over the chest prevented accurate positioning of the needle, a lateral roentgenogram of the chest was made (Fig. 1). This revealed the subcutaneous emphysema and the position of the needle within the pleural cavity. A needle was then inserted into the left chest and connected to a tube under water. Air likewise bubbled freely from this side. A catheter was then placed in the left pleural space because the air was coming more freely from this side. The air ceased to bubble from the right side so catheter drainage was not carried out.

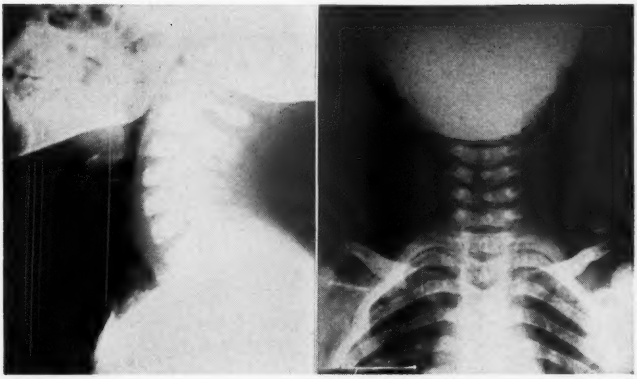


Fig. 3.—Antero-posterior and left lateral x-rays of the neck on the third postoperative day. Minimal amounts of air are in the fascial planes of the neck and absence of retro-tracheal thickening are demonstrated.

Specific treatment of the mediastinal emphysema and respiratory obstruction was considered at this time because the patient obtained only moderate relief from the aspiration and drainage of the chest. An antero-posterior roentgenogram of the chest was taken at this time (Fig. 2), but the subcutaneous emphysema masked the evidence of mediastinal emphysema which may have been present. A mediastinotomy was deemed advisable. A midline incision was made from the suprasternal notch to the lower border of the cricoid cartilage. The dissection was continued down to the anterior wall of the trachea and a considerable volume of air was released after the strap muscles were separated in the midline. A tracheotomy was considered but at this time the dyspnea appeared to be sufficiently alleviated so it was deemed unnecessary. The wound was packed with dry gauze.

The postoperative course was uneventful. Antibiotics consisting of penicillin and dihydrostreptomycin in adequate dosages, were administered. The tube was removed from the left pleural space on the second postoperative day. The pack in the cervical mediastinotomy incision was changed on the first postoperative day and removed on the second postoperative day. The patient ran a low grade fever on the first and second postoperative days reaching a maximum of 100.2 F. The subcutaneous emphysema gradually absorbed (Fig. 3 and 4) and had entirely disappeared at the time of his discharge from the hospital on the ninth postoperative day. When

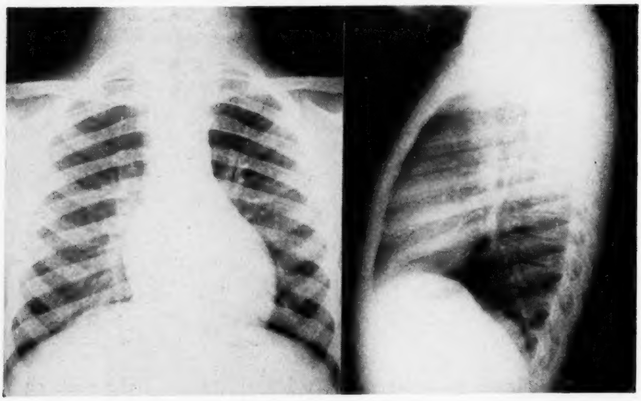


Fig. 4.—Left lateral and antero-posterior chest x-rays on the ninth postoperative day. The emphysema has absorbed and the lung fields have returned to their preoperative condition.

last observed, four months after the bronchoscopic procedure, the child evidenced no ill effects.

DISCUSSION

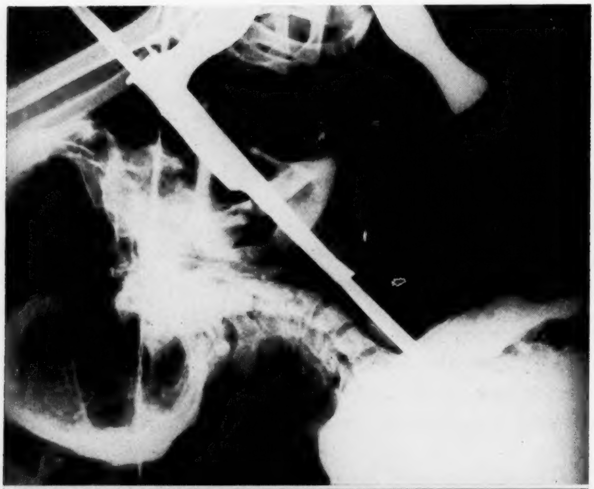
The extreme movement of the shoulders and head, just after introduction of the small size bronchoscope was the mechanism of the rupture of the trachea. The child was of the hyperkinetic type and had not been cooperative at the initial examination in the bronchoscopic out-patient clinic. It is not surprising that the child became unruly in spite of the usual premedication. General anesthesia should be considered for patients of this type.

The "heaving" movement of the head and shoulders resulted in an up and down motion of the head in relation to the chest with the bronchoscope maintaining a constant angle with the horizontal. The usual movement is a combined lowering of the head and rotation about a theoretical axis at the entrance to the chest (Fig. 5). The failure of rotation in this case resulted in a lengthening and shearing action of the end of the bronchoscope on the trachea as diagramed in Fig. 6 and 7. The relatively small size bronchoscope applied this strain over a small area and the rupture resulted. The tear in the trachea was undoubtedly transverse and between tracheal cartilages. When the subcutaneous emphysema occurred, it was assumed that there was mediastinal emphysema and that there was the possibility of a unilateral or bilateral pneumothorax and respiratory obstruction. The child's serious condition did not allow time for roentgenological examination and the swelling of the chest prevented adequate physical examination. Observation of venous engorgement was not carried out.

The treatment of such a complication consists of "100%" oxygen administration, chest aspiration, mediastinotomy and tracheotomy. The administration of "100%" oxygen makes the gas that escapes into the tissues more readily absorbed than if air containing 79% nitrogen were breathed. The oxygen further helps to reduce any anoxia that might be present. Aspiration of the chest was a simpler and quicker procedure than mediastinotomy or tracheotomy so it was done first. When the pneumothorax was definitely diagnosed, the opposite chest was aspirated. The catheter was inserted in order to allow continuous escape of the air from the left side and was thought unnecessary on the right side.

The treatment was continued by proceding with the mediastinotomy. This was performed in the mid-line so that a tracheotomy tube could be inserted if it became necessary. The escape of air from the wound after the strap muscles were separated produced considerable relief of the dyspnea and a tracheotomy was not considered essential. No evidence of the rupture was observed in the anterior wall of the trachea in the area exposed during the mediastinotomy. The site of the rupture was probably below this area and involved the right lateral and posterior wall.

The mechanisms involved in the production of mediastinal emphysema is first the difference in pressure within the trachea and the mediastinum, plus a valvular action at the site of the rupture. During inspiration, air passes into the mediastinum because the negative pressure there, is greater than in the trachea. During expiration, air passes from the mediastinum into the trachea because the positive pressure is greater in the mediastinum than in the trachea. The valvular action, related perhaps to the constriction of the trachea and reduction of intrathoracic volume during expiration, results in the air being trapped in the mediastinum. The resulting accumulation of pressure within the chest produces respiratory embarrassment and increased respiratory effort. This explanation of mediastinal emphysema is in general accord with the concepts of Macklin;[1] Maness[2] and Hammond[3] in association with bronchial foreign bodies and their removal; Sheinfeld,[4] in relation to chest trauma, and Meek[5] in consideration of the problems of surgery of the neck.



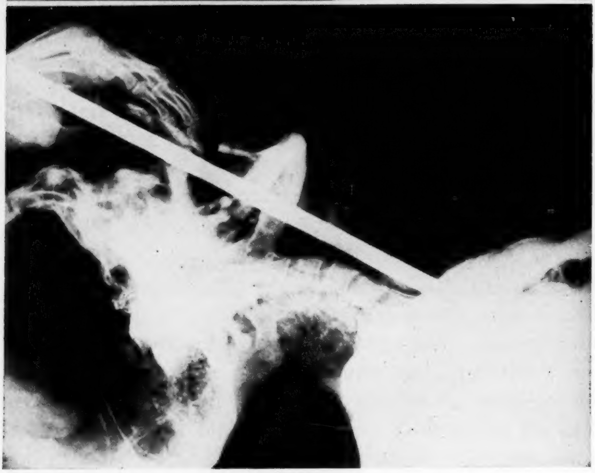


Fig. 5*a*.—Lateral x-ray (cadaver) demonstrating positioning for the introduction of the bronchoscope through the laryngoscope.

Fig. 5*b*.—Lateral x-ray (cadaver) demonstrating positioning for passage of the bronchoscope through the distal trachea.

The air which infiltrated through the tissues and passed into the pleural space, escaped through a site which is unknown. There was the possibility, of course, that the air passed from one pleural space into the other, rather than by a bilateral passage of air into both pleural spaces. The rather rapid cessation of the escape of air from the right chest after the catheter was placed in the left pleural space suggests such a mechanism. Karns and Dane[6] contend that the occurrence of subcutaneous emphysema relieves the pressure of the mediastinal emphysema and acts as a safety valve. The diagnosis of mediastinal emphysema and mediastinal tamponade could not be recognized by the usual signs, consisting of crepitation over the precordium synchronous with the heart beat (Hamman's sign[7]), of venous engorgement, an increase in the difference in systolic blood pressure during inspiration and expiration, and roentgenographic evidence of an extensive amount of air in the mediastinum. Howover, the persistence of dyspnea after relief of the bilateral pneumothorax in the absence of obstructive signs, was considered presumptive evidence of high mediastinal pressure.

Positioning of the patient for peroral endoscopy requires not only the cooperation of the head-holder in association with the endoscopist, but the endoscopist with the head-holder as well. The disregard of the importance of the latter is one of the principle causes for unsatisfactory positioning. In the first phase of this cooperative association, the endoscopist indicates to the head-holder and shoulder-holder by word of mouth or a previously agreed upon signal, the refinements of the position of the head which are required for the individual case during the entire procedure. It is assumed, of course, that the head-holder is experienced and understands the principles of endoscopy. If the head-holder is inexperienced, more understanding cooperation on the part of the endoscopist is required and if his instructions are adequate, adequate head-holding results. The second phase in the cooperation of the endoscopist and the head-holder consists of the initial assistance in placing the head in the proper position. The endoscopist employs his right hand, placed on the occiput and over the hand of the head-holder when the Jackson technique is used, in order to flex or to extend the head or to make any combination of these two movements. This approach of the endoscopist is such that he can control the force being applied more adequately than can the head-holder. This maneuver by the endoscopist may be used when the correlation of lowering and rotating of the head is proceeding in an unsatisfactory manner. In the case reported, the accident might have been prevented had the endoscopist assisted the head-holder by placing his right hand on the occiput and aided in steadying the head.

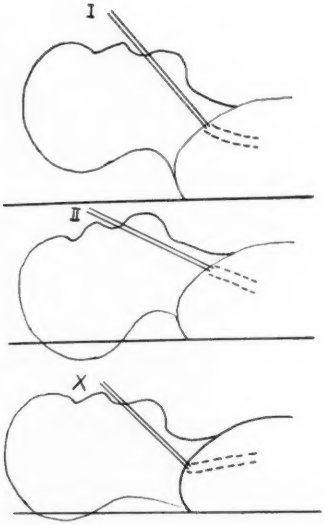


Fig. 6.—Diagram of positioning for bronchoscopy. I and II are made from Fig. 8 and 9 and illustrate the lowering and rotation of the head and show the stretching and bending of the trachea.

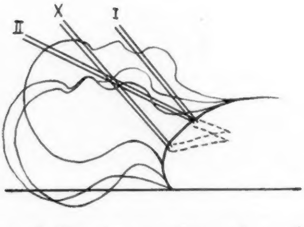


Fig. 7.—Diagrams of Fig. 10 superimposed to demonstrate the unsatisfactory position at X as compared to the satisfactory positions and I and II.

SUMMARY

A case of rupture of the trachea occuring during bronchoscopy on a seven and one-half year old child; medical and surgical management resulted in complete recovery.

The principal cause of the rupture was the uncontrolled movements of the patient's head and shoulders after the introduction of the bronchoscope subsequent to the removal of the laryngoscope. The ruptured edge of the trachea and slight bleeding were immediately observed and the procedure was terminated. Mediastinal emphysema massive subcutaneous emphysema and bilateral pneumothorax ensued.

Specific treatment consisted of "100%" oxygen administered by mask, bilateral pleural cavity aspiration and drainage, and cervical mediastinotomy.

Stress is placed on the importance of evaluation of the patient in terms of his controllability during bronchoscopy without the employment of general anesthesia.

The correlated lowering and rotation of the head has been demonstrated by roentgenograms and schematic drawings; the failure of rotation was the principal cause of the rupture in the case which is the subject of this report.

The responsibilities of the endoscopist in positioning of the patient consists of directions to the head-holder and shoulder-holder and his own manual assistance to the head-holder as may be required. These are discussed in detail.

104 South Michigan Boulevard.

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DISCUSSION

Dr. Paul C. Samson (Oakland, Calif.): We have all listened with great interest to this excellent presentation by Dr. Andrews. I, likewise, wish to record a case of airway rupture during endoscopy. This was performed in the army but unfortunately, I do not have x-ray proof at hand, however I can assure you that the tear was there. This young fellow was 20 years of age, rather a 5 x 5, with large shoulders, short neck and jutting jaw, one who is approached with some trepidation insofar as endoscopic procedures are concerned. His reason for endoscopy was a continuing cough and repeated colds. He wanted to know whether he was going to be a fit subject for the military or for a CDD.

The 8x40 bronchoscope was passed directly. The first part of the examination went without difficulty. The left stem bronchus was at a little more of an angle than usual from the trachea and we passed into the left stem bronchus, the patient responded to our effort with a violent cough and forcibly turned his head from the right shoulder to the midline. We grabbed wildly at teeth and gums and got his head back. I looked down and saw what appeared to be a very much injured mucosa. We went ahead and aspirated the secretions and since we were going to do a bronchogram and since the man seemed to be carrying on fairly well, we made a fluoroscopic examination with lipiodol that showed a trickle of barium in the mediastinum. We went back to the ward and I told the ward surgeon that very shortly he would complain of swelling of the face. Two hours later I was called and, as Dr. Andrews has said, the swelling as reported by the ward surgeon was an understatement. We made preparations to do a mediastinotomy and perhaps go in and repair the left stem bronchus, but first thought we would try observation. We sedated him greatly and placed him in an oxygen tent. Luckily for us, I believe, within two days there was definite evidence of regression of the mediastinal and subcutaneous emphysema. He went on to recovery without surgery being necessary.

There is one point I would like to bring up in discussion of Dr. Andrews' paper and that is his mention of the possibility of tracheotomy. In the case of tracheal rupture, I think that probably a tracheotomy should be considered, not because we are having airway obstruction, but for the reason that air being forced into the mediastium is forced against a closed or partially closed glottis. If a tracheotomy is performed, that will bypass the closed glottis and will, I believe, make less of a passage of air into the mediastium and thence of course into the pleural cavities.

We had experience with four or five cases of neck wounds during the war in which the trachea was creased or perforated and it seemed to us that tracheotomy was a good procedure to do early.

It was a pleasure to listen to this case and I think the essayist should be congratulated.

Dr. Shirley H. Baron (San Francisco, Calif.): I was glad to hear Dr. Andrews mention at the end of his paper that general anesthesia as a prophylactic measure against rupture of the trachea should be given consideration. I should like to state that most of us in this part of the country abandoned local aneshesia for bronchoscopy in children. We are doing our bronchoscopy in children under general anesthesia. We have had no reason to regret it. We do not have to worry about a head-holder. We have the complete cooperation of the patient without psychic trauma and without bad after-effects.

Dr. Albert H. Andrews, Jr. (Closing): Time does not permit a discussion of the use of general anesthesia for bronchoscopy in children. Varying views exist on this subject but it is interesting to note that this case is the only complication of bronchoscopy that has occurred in the thousands of procedures that we have done since 1935.

The mechanism of production of mediastinal emphysema following rupture of the trachea is not well understood. It would seem that the activating force is the differential pressure between the mediastinum and the trachea. This would produce a to and fro movement of air but the element of a valvular block occurs so that air cannot return into the trachea.

The extreme positive pressure which occurs has a result of respiratory effort forces the air into the neck to produce the subcutaneous emphysema. This mechanism may be different from that which produces mediastinal and subcutaneous emphysema after rupture of pulmonary blebs or in the presence of bronchial foreign bodies.

LXXII

TRACHEOBRONCHIAL TOILET IN INFANT AND ADULT

Paul C. Samson, M.D.

and

David J. Dugan, M.D. (By Invitation)

Oakland, Calif.

The necessity for continuous tracheobronchial patency in spite of injury and disease needs repeated emphasis, particularly among physicians not habitually thinking in terms of a clear airway. Unfortunately, it is not unknown for the attending physician to conclude with regret that he must passively accept the impending catastrophe of obstruction. While its importance had been recognized by many of us for a number of years, experiences during the recent War served to intensify our respect for the lethal potentialities of impaired tracheobronchial drainage.

In this report, we are concerned only with an airway which is structurely normal. The offenders are fluid substances of various kinds and from various sources which clog the passages and obstruct the orifices. The fully conscious patient with painless chest and an efficient cough mechanism may rid himself of these noxious fluids unaided. Under a variety of circumstances, however, mechanical help is necessary.

Bronchoscopy has long been, and still remains the most useful adjunct in such situations. The advantages need not be detailed before this audience. There are very real disadvantages, however, which make advisable the availability of simpler techniques which can be readily taught. These techniques differ somewhat in infant and adult. The disadvantages of bronchoscopy may be briefly listed: the need for special equipment, anesthesia and a trained endoscopist; the necessity for repeated aspirations in many cases.

TRACHEOBRONCHIAL ASPIRATION IN THE INFANT

In the infant, bronchoscopy carries slightly more risk than in the adult and, since many of the patients needing relief are premature, the hazard is even greater. Since one is unable consistently to

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Presented at the 31st Annual Meeting of the American Broncho-Esophagological Association, May 25-26, 1950 at San Francisco, California.

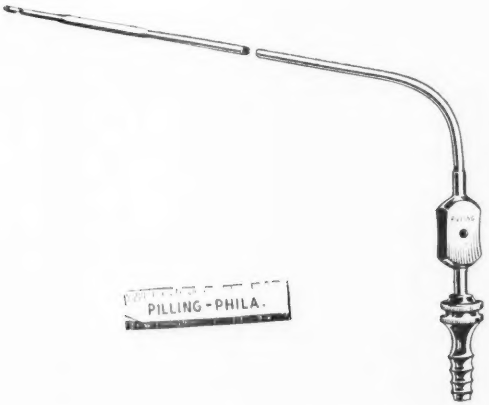

Fig. 1.—Samson-Davis Tracheobronchial Suction Tube. It is composed of small-bore metal tubing. On the distal tip there is cemented a segment of No. 8 F. rubber catheter with end and side holes. The proximal thumb control is necessary for intermittent suction.

pass a catheter blindly in the infant, the larynx must be exposed directly. A catheter of a size which can be safely used is too limber to be passed between the cords with efficiency, and a special suction tube has been devised (Fig. 1).

Technique: The infant is mummified and direct laryngoscopy is performed. The hypopharynx is flooded with oxygen after the suggestion of Andrews[1] (Fig. 2). Without suction, the special aspirating tube is rapidly advanced to its fullest extent by gentle pressure. This places the tip in the inferior portion of the right stem bronchus. The tube is withdrawn into the trachea while applying rapid intermittent suction with the thumb control. The baby will cough violently, strangle and may become cyanotic. Under any circumstance, the procedure should consume only a few seconds. Suction may be repeated on the right and the tube temporarily withdrawn. To enter the left bronchus, the head is turned to the right and the lip of the laryngoscope to the left. The soft tip of the suction tube is advanced carefully along the left lateral tracheal wall.

Advantages: Suction of this type may be repeated at hourly intervals. Pediatric residents can be taught the technique without too great difficulty. The equipment is simple and can be made readily available on the wards.

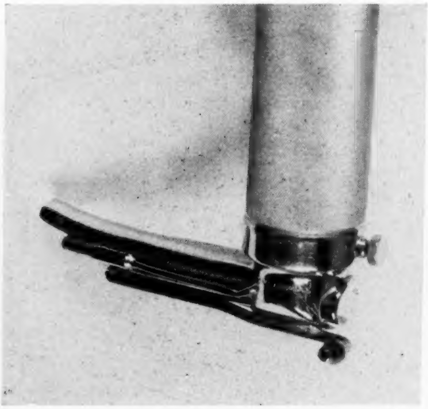

Fig. 2.—Welch-Allyn Direct Laryngoscope fitted with special infant blade. A side channel has been added (Davis modification) for the continuous administration of oxygen.

Disadvantages: The procedure is blind. Laryngeal spasm is a possibility, but it has not been troublesome in our experience.

Indications: The prime indication is that of excess secretion. We emphasize that the infant has a poorly developed cough mechanism and cannot help itself. Frequently, newborn infants will have aspirated amniotic fluid and meconium or will show evidence of over-abundant mucus production. Babies with swallowing difficulties (congenital tracheo-esophageal fistula, esophageal stenosis, cicatricial stricture) may have a spill-over into their bronchial tree necessitating frequent aspirations. A wet "soupy" tracheo-bronchitis may be an indication. During the resolution stage of broncho- or lobar pneumonia, secretions may flood the bronchi and trachea. Some patients develop tracheobronchial wetness following operation.

Signs and Symptoms: Most of these infants have some degree of anoxia. They are either listless or very restless. Respirations are rapid and cyanosis may be present. One important sign is labored retraction of the lower costal arch and xiphoid on inspiration (Fig. 3). This is presumably due to diffuse partial bronchial obstruction. Physical examination shows coarse, bubbling rales over the lung fields and trachea. Oral rhonchi are obvious. The roentgenogram may be relatively non-informative and frequently alarming signs may be out of proportion to roentgen findings. At times, patchy

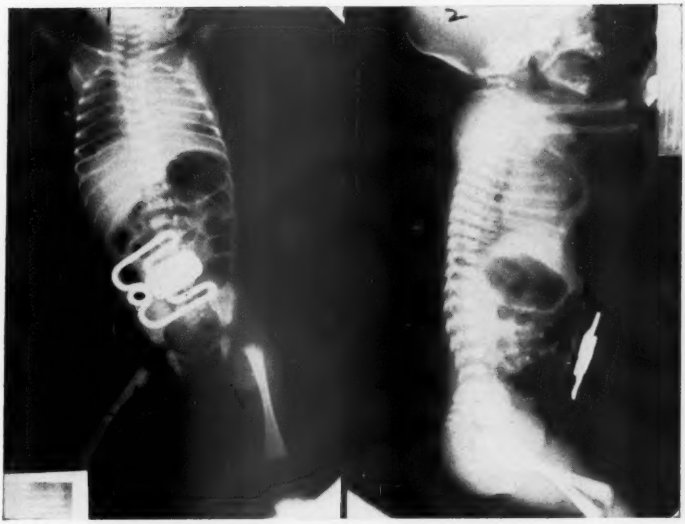

Fig. 3.—Roentgen films taken shortly after Cesarean delivery of a premature infant girl weighing 4 lbs., 7 oz. The frontal view contributes little. Note marked indrawing of the costal arch in the lateral projection. The baby breathed poorly from the first and was persistently cyanotic with rapid, shallow respirations. Coarse rales were heard throughout both lung fields. The first aspiration, performed 1½ hours after birth, resulted in dramatic improvement. Four more aspirations were necessary in the following 36 hours. Gavage feedings were employed for the first four days. There was complete recovery.

areas of increased density, segmental and lobar atelectasis, or expiratory check-valve mechanisms (Fig. 4) may be seen.

Ancillary Treatment: Hydration should be maintained but parenteral fluids should not be given rapidly or in large amounts because of the danger of pulmonary adema. Gavage feedings for several days have helped to lessen the burden on a low pulmonary reserve. Increased depth in respiratory movements is sometimes valuable with forced expiration and crying initiated by sharply spanking the soles of the feet. Continuous oxygen is always given. A moist environment is necessary. The recent development of a completely aerosolized atmosphere[2] seems to be of value. Under such a situation, antibiotics can be easily and continuously administered. We have routinely employed penicillin intramuscularly as prophylaxis.

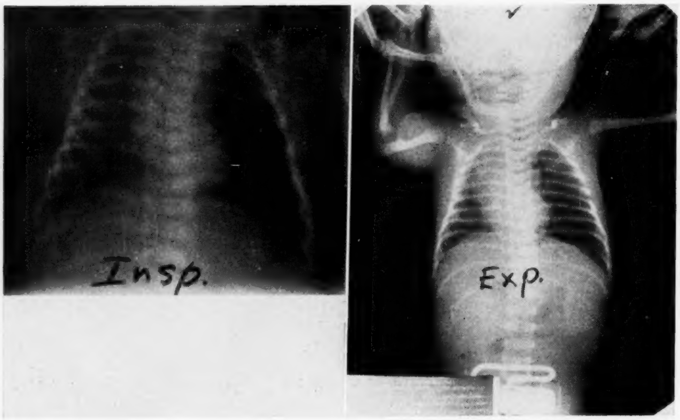

Fig. 4.—Roentgen film taken aproximately 8 hours after full-term normal delivery. There is an expiratory check-valve mechanism with obstruction of the left main bronchus. There had been no immediate respiratory difficulty. Gradual onset of restlessness, and the infant seemed to be struggling to breathe. There was slight cyanosis unrelieved by oxygen. The breath sounds were absent on the left. The condition gradually deteriorated. Tracheobronchial suction 12 hours after birth was productive of thick, tenacious mucus. Fluoroscopy immediately following suction showed a normal chest.

TRACHEOBRONCHIAL ASPIRATION IN THE ADULT

In the adult there are two common indications for catheter suction. In traumatic wet lungs excessive secretions, purulent exudate, blood and edema fluid may flood the air-way and necessitate repeated aspirations[3-5] (Fig. 5). In many post-operative patients (particularly following pulmonary resection for suppuration) retained secretions must be removed. Less frequently catheter suction may be necessary at the end of an operation if the indicated bronchoscopy is impossible because of partial reaction from anesthesia. Following *primary* bronchoscopy for aspiration "pneumonitis," a residual chemical bronchorrhea may be handled by catheter suction.

Signs and Symptoms: The cardinal signs and symptoms of retained secretions should be recognzed. The cough is "wet" and may be continuous, harassing, hacking or paroxysmal in character. The expulsive force is weak. The patient wheezes and rattles in spite of raising small amounts of sputum. The very fact that the cough is continual even though slightly productive, is pathognomonic of inadequate bronchial drainage. Dyspnea is almost always present.

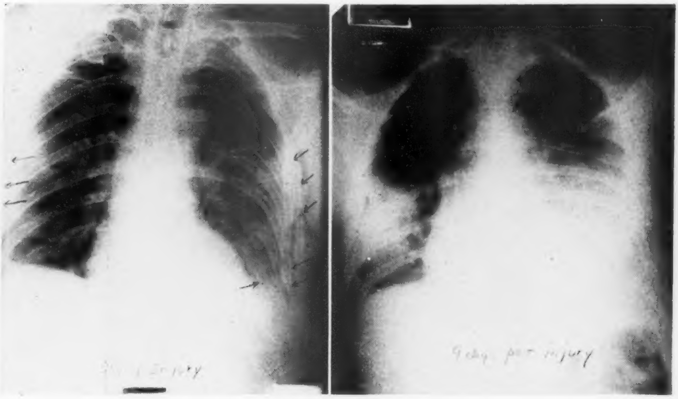

Fig. 5.—Traumatic wet lung in a male, aged 58.

A. Portable film, day of injury. There are bilateral pulmonary contusions, bilateral multiple rib fractures and left-sided hemothorax. Rapid deterioration 3 days after injury.

B. Portable film 9 days after injury. Still critically ill but intensive therapy continued. Rapid recovery shortly after this date. During one week's time the following therapy was given: Regional intercostal nerve block, left; thoracentesis, left; thoracentesis, right, three times; tracheobronchial catheter aspiration, 15 times; bronchoscopy, once; three blood transfusions.

Frequently, the patient is very restless or cyanotic, either of which is a sign of anoxia.

On physical examination there are oral rhonchi and bronchial râles. The râles are usually coarse and may have a dry quality or be bubbling and sonorous. Typically, râles are increased greatly and persist after cough and expectoration. In fact, they may be inaudible during quiet respiration.

Fever and toxicity are constantly present. While early infection is a minor factor, the fallacy of treating these patients solely for bronchopneumonia should be obvious. The history, the constant wet cough and widespread bronchial râles are sign posts that will not be ignored in the proper evaluation of the patient's condition.

Technique: In the adult it is almost always possible to pass a rubber tube blindly into the tracheobronchial tree. Several technical variations have been described.[6-9] With slight modification,[10] we have routinely employed the technique originally described by Haight.[6] The necessary materials consist of a new No. 16-18 F. catheter of the open end or "whistle tip" type, connecting tubing

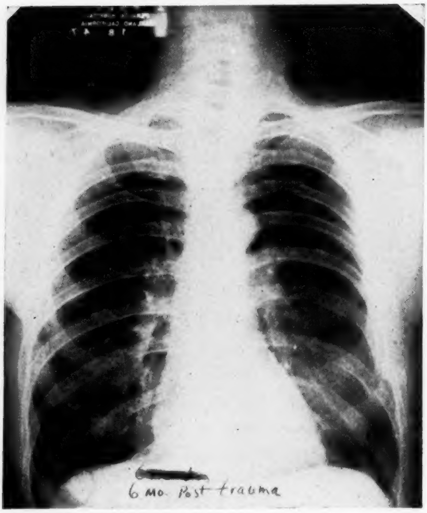

Fig. 6.—Roentgen film 6 months later showing complete clearing of chest.

(Reprinted from Samson, P. C.: The Care of Thoracic Injuries, Arizona Med. 4:32 (Sept.) 1947.)

and an electrical suction machine capable of delivering 15 pounds of negative pressure. Two additional small holes are cut in the distal two inches of the catheter. Many times, the catheter can be passed more easily if the distal end is angulated (Fig. 7). The catheters should not be repeatedly autoclaved and should be kept uncurled when not in use.

No anesthesia is necessary. The patient is placed in a semi-upright position and the neck is flexed sharply forward. If possible, other tubes should be removed from the nose. The tongue is pulled sharply forward to elevate the epiglottis. Without suction, the catheter is passed through the more patent nostril to the larynx. It is then rapidly advanced during either deep inspiration or the expiratory phase of a cough. This manuever may have to be repeated several times. If the patient retches or gags, the catheter has not passed the vocal cords. Success in entering the trachea will be signified by hoarseness of the voice, stridor, and violent coughing. The catheter is advanced to its full length which puts the tip in the depths of the right stem bronchus. Suction is then applied intermittently for not more than from three to five seconds at a

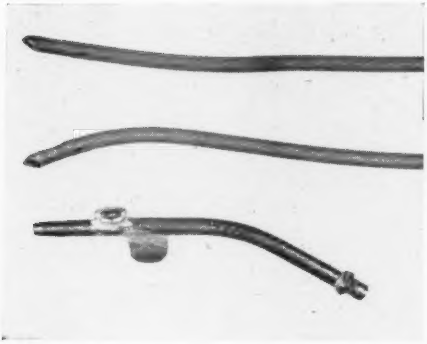

Fig. 7.—Straight and angled catheters of open-end type, No. 16-18 F. which are eminently satisfactory for tracheobronchial aspiration. Note the extra holes. The lower catheter was threaded on a stylette with a 30 degree angle in the distal 2 inches. This has been autoclaved for 10 minutes at 10 lbs. pressure to preserve the angle. The metal connecting tube has a convenient finger-tip control for intermittent suction.

time, while the catheter is being slowly withdrawn into the trachea. The patient will continue to cough violently and may become slightly cyanotic. Suction is discontinued and oxygen at four liters per minute passed directly through the catheter. After thoroughly aspirating the right stem bronchus and the trachea, the chin is sharply elevated, the head turned well to the right and the catheter again advanced. In most instances, it will enter the left stem bronchus. The process of intermittent suction should be repeated. In the unconscious patient a finger may be introduced directly through the mouth to engage the epiglottis. In such a situation, however, it is probably better to pass an endotracheal tube as described by Foregger.[9]

In a small percentage of patients, the anatomy of the epiglottis is such that the catheter cannot be passed blindly. In such situations, the catheter may be passed through a direct laryngoscope, working from the front or side with the patient in semi-Fowlers position. The oral route is likewise necessary if both nostrils contain necessary tubes. The patient may be turned with his worse side uppermost so that gravity drainage aids in loosening secretions from the smaller bronchi. The catheter may be left indwelling between aspirations and oxygen administered through it at four liters per minute.

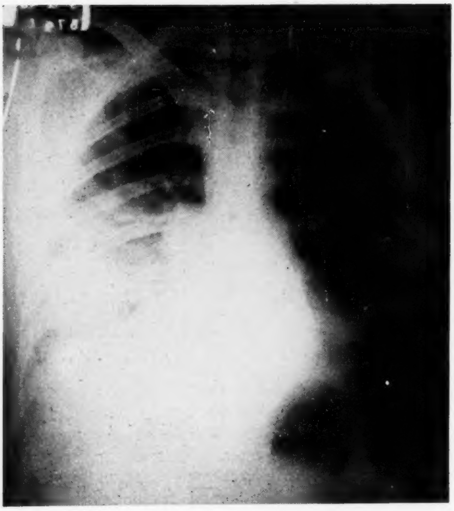

Fig. 8.—*A*. Roentgenogram of a soldier three days after wounding. There is bilateral basal atelectasis and right-sided pulmonary contusion. The patient was dyspneic, exhausted, dehydrated and his cough had no expulsive force. This was considered an indication for primary bronchoscopy.

Rarely, as with a fractured jaw, neither bronchoscopy nor catheter suction is possible. Under these circumstances, tracheotomy is a perfectly justifiable procedure to permit access to the air-way.

Advantages: With some practice, catheter aspiration can be learned very easily. The materials are simple and readily obtainable. No anesthetic is needed. The procedure can be performd evry two or three hours if necessary.

Disadvantages: Catheter aspiration is not as certain as bronchoscopy. It is a blind procedure and it is not efficient in removing inspissated material, clotted blood, or particular matter. In any event we resort to bronchoscopy immediately if catheter suction fails to relieve symptoms.

Ancillary Measures: Oxygen usually should be administered. Intermittent carbon dioxide inhalations may aid in evacuating secretions but should not be given if the patient is cyanotic. A wet atmosphere is often helpful. Positive pressure oxygen and, rarely, atropine may be used if an element of pulmonary edema can be detected.[3, 4] Intercostal nerve block will relieve thoracic pain and

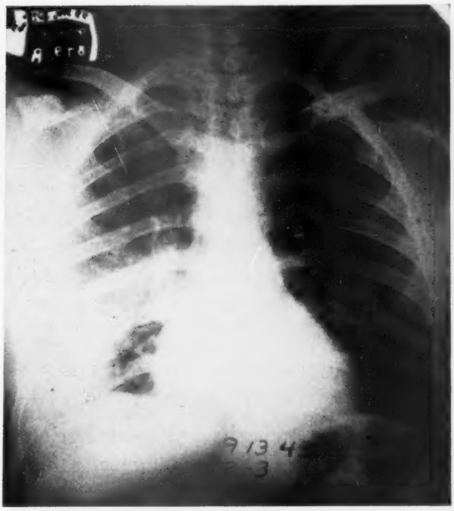

Fig. 9.—Twelve hours following bronchoscopy. The atelectasis has cleared and the contusion remains. Prior to this film the patient raised six ounces of thick, sanguineous sputum containing many small plugs. (Reprinted from Samson, P. C., and Brewer, L. A.[5])

the patient will thereafter be able to cough more efficiently. Air or fluid in the pleural cavity should be promptly aspirated. Morphine should be used sparingly. Penicillin is employed almost routinely, both by the aerosol and intramuscular routes.

INDICATIONS FOR PRIMARY BRONCHOSCOPY

In the adult particularly, a primary bronchoscopy may be indicated under certain conditions: (1) for inspissated secretions; (2) for blood clots; (3) for suspected particulate matter, as in the aspiration of stomach contents; (4) for tracheal obstruction (5) for pulmonary atelectasis which is lobar or greater in extent (Fig. 8). A patient who is completely exhausted, without the strength to cough and whose air-way is brimming with secretions should have bronchoscopy rather than be aspirated with a catheter. We emphasize that no patient is too ill to be bronchoscoped. It is a good general rule that the sicker and more anoxic the patient, the more necessary is immediate bronchoscopy. In situations of this sort Williams[11] prefers endoscopy without any anesthesia. When an early operation is mandatory in the face of persisting tracheobronchial wetness, bronchoscopy should precede the introduction of an endotracheal anesthetic tube.

SUMMARY AND CONCLUSIONS

During the course of disease or following trauma, situations arise in which fluids of various types threaten the patency of the trachea and bronchi. If the patient cannot clear his own air-way by means of cough, mechanical toilet of the tracheobronchial tree will become necessary. Bronchoscopy with aspiration and mucosal shrinkage has long been recognized as the most efficient means of achieving this end. Under a variety of conditions, however, complementary and supplementary and supplementary procedures are advantageous.

The varying techniques of these procedures have been presented as they apply to the infant and to the adult.

The indications for tracheobronchial aspiration have been outlined and the relationships of these maneuvers to bronchoscopy have been discussed.

Ancillary measures of value have been listed.

The indications for primary bronchoscopy have been given.

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DISCUSSION

Dr. David J. Dugan (Oakland, Calif.): I have very little to add to this discussion except to emphasize the amazing incidence we have found of aspiration of the secretions in the infant and the adult. The use of this tracheobronchial suction has been brought out very clearly in the treatment of postoperative lobectomies. While the majority of the men here present are not engaged in this type of work, we feel that we have gotten a great deal from the endoscopists and the broncho-esophagologists on the clearing of the tracheobronchial tree following lobectomy. This has been advantageous in pneumonectomy because we like to withhold bronchoscopy if possible on these patients who are acutely ill. Using the tracheobronchial suction has avoided that procedure considerably.

Dr. Paul H. Holinger (Chicago, Ill.): The paper by Dr. Samson and Dr. Dugan is of great interest. I think this subject was stimulated again by a recent article in "The Journal of The American Medical Association" which brought to light the fact that the internist has finally discovered a procedure which has been used by generations of broncho-esophagologists. Dr. Samson alluded to the excellent work of Dr. Cameron Haight in his presentation, and this is of great importance. The Samson Aspirator, which Dr. Samson didn't present at this time, however, is one which we find almost indispensable in the management of the respiration obstructions in children. It is easy to use and a most satisfactory instrument for this problem.

In discussing the problem of tracheobronchial aspiration in infants, it is of importance to stress the value of the x-ray prior to the procedure. Dr. Samson did not mention whether or not routine chest x-rays are taken of every infant prior to aspiration, and I believe the enthusiasm that sometimes follows reports such as his leads to use of the procedure without proper or complete study of the infant first. The danger of trauma or the danger of injury to an infant by aspiration procedures must also be considered and the recognition of some of the contra-indications is the reason for insisting on the x-ray prior to the aspiration. In an infant with a wet bronchial tree that requires aspiration, the most common findings we have noted is that the infant has had a brain injury during delivery, is vegetative, and the pharyngeal secretion cannot either be coughed up or swallowed. Pharynx aspiration in such an infant may be of importance but one has to be guarded in endoscopic procedures for fear of increasing cerebral hemorrhage. In such cases, postural drainage together with simple pharynx aspiration may be of assistance.

In one of our cases of newborns referred for aspiration because of cyanosis, dyspnea and rales, x-ray demonstrated a spontaneous pneumothorax. In another case of a newborn infant, the presence of a large tension lung cyst was detected. In both cases bronchoscopy was contra-indicated. Dr. Samson is familiar with these; I mention them because of the importance of x-ray prior to the endoscopy tracheobronchial toilet.

Dr. Gabriel Tucker (Philadelphia, Pa.): I wish to compliment Dr. Samson on his presentation. The importance of a clear airway in both infants and adults postoperatively cannot be overemphasized. It is my belief that the surgeon and assistant or the anesthetist in charge should be trained in the technic of examination of the larynx and tracheobronchial tree so that the area may be inspected without trauma to either the infant or adult.

This visual examination should be made when the first symptoms are manifest. Blind introduction of an aspiroator where there is foreign material in the tracheobronchial tree is inadvisable, especially in an emergency procedure. When a laryngoscope is introduced the pharynx and larynx are visualized and obstructive material removed without trauma. Paralysis and laryngeal disease are revealed or excluded by this examination. The bronchoscope is introduced without trauma and the tracheobronchial tree is inspected in the same manner. With the introduction of the bronchoscope, an airway is established allowing oxygen insufflation while obstructing material is removed. This technic is easily acquired and the knowledge obtained should form the logical basis for the further "toilet" of the tracheobronchial tree. If further aspiration is required, the introduction of an aspirator through a direct laryngoscope with a flow of oxygen into the larynx may be justified without the use of a bronchoscope. The procedures of direct examination of the larynx and tracheobronchial tree can be carried out without anesthesia in the infant and with local anesthesia in the adult. It is desirable that the individuals who are responsible for the care of postoperative cases should practice these procedures on the cadaver and living dog before they assume the responsibility for human lives.

If secretions accumulate, following the clearing of the tracheobronchial tree, the introduction of an aspirating tube into the pharynx without anesthesia will usually excite sufficient cough to clear the trachea and bronchi. This procedure may be delegated to a competent nurse. Laryngotracheobronchial complications, however, in thoracic surgery and otolaryngology are of sufficient importance to justify the limited training in bronchology and the instrumentarium required to visualize the larynx and tracheobronchial tree. These procedures can then be done more safely and efficiently.

The importance of roentgenologic diagnostic studies prior to any type of manipulation in postoperative pulmonary complications cannot be over emphasized. Roentgenologic examination will usually determine if "toilet" of the tracheobronchial tree is required.

DR. PAUL C. SAMSON (closing): I wish to thank my colleague, Dr. Dugan and Drs. Holinger and Tucker for their constructive remarks.

In answer to Dr. Holinger's query regarding x-ray, we certainly take x-rays, I would say practically routinely. Perhaps not 100% because sometimes it seems so obvious that immediate help is necessary that we do not want to take the time to take the baby upstairs to get an x-ray. That is infrequent however and his remarks concerning the value of the preaspiration or prebronchoscopic x-ray are well taken and I agree perfectly.

I agree too, Dr. Tucker, that we like to look. I offer this procedure not so much for this audience perhaps as for those who cannot do bronchoscopies and in regions where bronchoscopy is not available. Believe it or not, there are small sections of the country where that is true. If he will recall my remarks concerning the infant and the type of suction we do in the infant, we do look at the cord directly and aspirate or pass our small tube through the laryngoscope. That is not a blind procedure insofar as laryngoscopy is concerned.

We have not had evidence of trauma following introduction of an 18 French catheter into the adult larynx and into the bronchial tree. Dr. Tucker is afraid that we will push the catheter through a freshly closed bronchus. That may be a theorectical disadvantage, but Dr. Tucker will have to take our word for it that we have done catheter suctions on many occasions following resection with no evidence of bronchial rupture. For those of you who do not know, Dr. Dugan and I are thoracic surgeons, not primarily bronchoscopists, so we are working on our own patients.

The necessity for repeated aspiration I believe is one good reason for the use of a tracheobronchial catheter suction. By repeated aspiration, I mean every two or three hours. Granted that one can do bronchoscopy twice a day, perhaps three times a day, I have never done it that frequently and I doubt very much if anyone wants to do a bronchoscopy every three hours day and night when the catheter suction can be used and seems to be quite efficient in many cases. Thank you very much.

LXXIII

OBSERVATIONS ON THE ESOPHAGUS FOLLOWING VAGOTOMY

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Current interest in the operation of vagus nerve section for the treatment of intractible peptic ulcer is widespread. Attending this revival have been renewed investigations in allied fields, notably anatomy and physiology. The esophagus figures prominently in this connection by virtue of its being the site of operative intervention. Quite naturally problems have arisen as to just what effects, if any, might be expected from interference with the nerve supply to this organ, or whether the procedure in any way alters normal esophageal function. This report will deal briefly with the anatomy and physiology of the vagus nerve but will be chiefly concerned with the symptomatology and esophagoscopic observations in a series of 31 ulcer patients who had undergone vagotomy for the treatment of chronic refractory peptic ulcer.

ANATOMIC CONSIDERATIONS

Standard texts of anatomy refer to the vagus nerve supply of the esophagus in both the superior and posterior mediastina. In the posterior mediastinum the esophagus is surrounded by the esophageal plexus, formed from the trunks of the vagi emerging from the posterior pulmonary plexuses. This part of the esophagus also receives fibres from the greater splanchnic nerve and ganglion (thoracic sympathetic system). Branches from the esophageal plexus supply the muscular wall and mucous membrane of the esophagus.[1] As a general rule it is stated that the major part of the anterior esophageal plexus consists of fibres from the left vagus nerve and the major part of the posterior plexus consists of fibres from the right vagus nerve.

From the esophageal plexuses are derived the gastric divisions of the vagus nerves; these are described as an anterior or left nerve trunk and a posterior or right nerve trunk, formed respectively from

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the anterior and posterior esophageal plexuses. (The distribution of the right vagus to the posterior surface of the esophagus and stomach and the left nerve to the anterior aspect of these organs is explained by the rotation on its right side of the originally symmetrically-situated stomach).

Several thorough investigations have recently been made on cadavers relative to the anatomic variations of the gastric divisions of the vagus nerve at the supradiaphragmatic levels. These have obviously resulted from attempts to further the completeness of vagus neurectomy and have added appreciably to the knowledge of the anatomy of this region.[2, 3, 4] The essence of this research may be summarized briefly as follows: In the majority of specimens examined (60% to 75%) there is a simple basic pattern whereby a single primary trunk arises from both the anterior and posterior esophageal plexuses, forming thereby the left, or anterior, and the right, or posterior nerves and entering the hiatus as single trunks. In the intermediate group, (16% to 22%) two or more secondary nerve trunks are formed from either of the single nerves before reaching the hiatus. A complex arrangement with multiple secondary branches or several distince primary trunks may be encountered in from 4% to 24% of specimens.

The practical application of these studies is to be found in the current preference for the transabdominal approach with supradiaphragmatic section of the vagus nerves along the lower esophagus.[5] To obtain the necessary exposure the peritoneum is incised over the esophageal hiatus and the esophagus is drawn into the abdomen, retraction being obtained with gauze tape or soft rubber drainage tubes. Approximately 6 cm of the esophagus can be visualized by this method and resection of the isolated nerve segments is carried out from 2 to 4 cm above the hiatus, before the nerves have had an opportunity to branch and enter the lower esophagus or stomach.[6]

In contrast to this procedure is the more extensive transthoracic neurectomy advocated by Moore in which there is removed a sizable segment of both nerves from the region of the lung root to the cardia of the stomach. This will be referred to later in considering the sequelae of clinical surgery.

PHYSIOLOGIC CONSIDERATIONS

The exact relation of the vagus nerves to gastrointestinal function is still obscure. At times the vagi seem to initiate functions in the manner of true motor or secretory nerves, while at other times they appear merely to augment or inhibit responses to local stimuli. These actions are referred to as phasic and tonic activities respectively. The results of animal experimentation vary so widely that no definite conclusions can be drawn from the mass of available data. Reputable investigators differ appreciably in their deductions on the effects of vagus stimulation, vagus section, the action of the sympathetic nerve supply and the nature of the cardiac sphincter. There is, however, one point upon which most physiologists seem to agree, namely that transthoracic or transabdominal vagotomy as usually performed on the human is seldom complete.[7] It is pointed out that branches of the esophageal plexus may be small and escape detection, that the gastric divisions of the vagus receive fibres from the esophageal wall above the level of operation and that vagus fibres may enter the esophageal wall and emerge again at lower levels.

In considering the effect of vagotomy upon the esophagus, two recent investigations merit attention. Ferguson subjected 10 monkeys to complete vagotomy and in all of the animals there developed striking evidence of cardiospasm, demonstrable clinically, roentgenographically and at autopsy.[8] He concluded that vagotomy removes the extrinsic reflex mechanism for the co-ordination of esophageal and gastric movements and for the control of the relaxation of the cardia. After vagotomy the cardia does not relax properly. Whether there exists a true spasm in addition to the failure of relaxation depends on the state of the local muscular tonus and this may vary from time to time and animal to animal.

Knight, experimenting on cats (because of the similarity of esophageal muscle content to humans) found that bilateral division of the vagi resulted in clinical symptoms and roentgenologic appearances of achalasia of a permanent nature. When the denervation was sufficiently severe, death of the animal resulted. Sympathetic denervation of the cardiac sphincter in the presence of induced achalasia produced complete cessation of symptoms and return of normal function roentgenographically.[9]

Thus in animals at least, operations that guarantee severance of all vagus fibres yield unfavorable results with respect to the esophagus.

REVIEW OF CLINICAL REPORTS

In a comprehensive review of the accumulated literature on vagotomy through 1947, Alvarez quoted no references to the esophagus.[10] Ruffin and White listed esophageal spasm as a complication of vagotomy.[11] They describe its transitory character and roentgenologic evidence of spasm of the entire lower third of the esophagus rather than true cardiospasm. The condition is portrayed as mild, subsiding spontaneously without the necessity of dilatation. Whether this is due to esophageal trauma at the time of operation or to a disturbance of the nervous mechanism is not known.

Suggestive evidence of cardiospasm in one patient following transabdominal vagotomy was reported by Ritvo and Shauffer[12] and after transthoracic vagotomy by Machella and Lorber.[13] In both patients the symptoms resolved spontaneously. Grimson and his associates noted dysphagia in 21 of 56 patients who had undergone transthoracic vagotomy.[14] This difficulty subsided in all of these patients and later roentgenograms were entirely negative. The authors concluded that achalasia had not occurred, as judged by negative roentgenologic examination and the failure of symptoms to persist.

Moore and his co-workers removed segments of the vagus nerve from near the lung root in the hope of severing all fibres which might enter the esophagus and later become expanded in their function to take over some of that lost by sectioning the main trunks.[15] Even with this extensive procedure no esophageal dysfunction was demonstrable.

Thus it appears that the literature to date conveys no indication of any permanent disturbances of esophageal function following either transthoracic or transabdominal vagotomy in the human. Dragstedt stresses that extensive esophageal resection for carcinoma and complete division of the vagus nerves do not lend to fatal impairment of function.[6] Again we note the discrepancy between animal and human response. Insofar as esophageal function is concerned, however, it is probably fortunate that a more complete vagotomy is not being advocated for humans.

ANALYSIS OF CASES

This study covers a group of 31 male patients who had undergone vagotomy and gastroenterostomy from 3 months to 3 years prior to investigation. In the majority, well over 1 year had elapsed since surgery. Because the survey has not been completed and since many of the patients returned because of persistent symptoms, it is not intended that these remarks be accepted as final judgment with respect to the esophageal sequelae of vagotomy. It should be mentioned parenthetically that relief from ulcer pain and distress has been claimed for 75% to 95% of patients undergoing this procedure.[6, 16]

In an effort to insure objectivity, tabulation of the patient's symptoms and the endoscopic examination were, as a rule, carried out by different individuals.

Symptoms Referable to the Esophagus—Pyrosis, substernal discomfort and eructation were most frequently noted, occurring in over 30 per cent of the patients. Regurgitation and dysphagia, though less common, were prominent complaints. Excessive saliva and hoarseness were noted to a lesser degree. (The latter symptom could not be attributed to intralaryngeal pathology or recurrent nerve paralysis in either of the three patients concerned.) (Table 1)

TABLE 1.
ESOPHAGEAL SYMPTOMS (POSTVAGOTOMY)
31 PATIENTS

Pyrosis	12
Substernal Discomfort	11
Eructation	10
Regurgitation	8
Dysphagia	7
Excess Saliva	5
Hoarseness	3

Roentgenologic Reports—Normal esophageal findings were recorded in 27 of the 31 patients examined (Table 2).

TABLE 2.
ROENTENOLOGIC REPORTS (POSTVAGOTOMY)
31 PATIENTS

Normal Esophagus	27
Hiatal Hernia	2
Achalasia	2
1 post-operative — transient	
1 intermittent — for 18 months	

Hiatal hernia was described in 2 patients and was probably a consequence of surgery. Achalasia occurred in 2 patients. In one of these it was demonstrated immediately postoperatively and disappeared in several days, no treatment being required. The other patient apparently had intermittent symptoms of achalasia for a period of 18 months after operation and persistent effort was required to obtain x-ray confirmation. This proof was finally forthcoming during one of his many fluoroscopic studies. The symptoms eventually disappeared entirely.

Esophagoscopic Observations—Excess secretion was encountered in 12 of the 31 patients examined endoscopically. Tests of this secretion on several of the patients revealed no free acid.

A patulous diaphragmatic hiatus was present in 6 patients. We are inclined to believe that this was a consequence of incomplete healing of the peritoneal incision over the diaphragm and that the hiatal hernias found in 2 patients had a similar basis.

Esophageal ulcer was observed in 2 patients. Benedict[17] and others have directed attention to the simultaneous occurrence of peptic ulceration in different portions of the upper digestive tract and increasing attention is being directed to the benign esophageal strictures which follow the healing of esophageal ulcers.

The average distance from the upper incisor teeth to the cardia in this series (all adult males) was found to be 40.3 cm, a figure which indicates the absence of any fibrosis or contraction of the esophagus following this particular procedure (Table 3).

TABLE 3.
ESOPHAGOSCOPY (POSTVAGOTOMY)
31 PATIENTS

Excess Secretion	12
Patulous Hiatus	6
Esophageal Ulcer	2
Average Distance to Cardia	40.3 cm.

Correlation of Symptoms and Esophagoscopic Findings—Despite the presence of symptoms, esophagoscopy proved entirely negative in some of the patients. On the other hand, positive findings were of definite interest when correlated with symptomatology. Attention is directed to the notation of excessive secretion in the esophagus in some patients who complained of pyrosis and substernal discomfort. The majority of those with eructation and excessive saliva were found to have an abnormal amount of secretion in the esophagus. Regurgitation and dysphagia are not well explained by the esophagoscopic picture but may conceivably be attributed to the intermittent esophageal dysfunction which reached a higher degree of activity in the patients with achalasia. One patient, symptom-free save for occasional tarry stools, was found to have an ulcer of the esophagus (Table 4).

TABLE 4.
CORRELATION OF SYMPTOMS AND ESOPHAGOSCOPIC FINDINGS
(POSTVAGOTOMY)—31 PATIENTS

SYMPTOM	NO. OF PATIENTS	NEGATIVE ESOPHAGOSCOPY	EXCESS SECRETION	PATULOUS HIATUS	ULCER
Pyrosis	12	5	4	3	
Substernal Distress	11	6	4		1
Eructation	10	3	6	2	
Regurgitation of food	8	5	3	2	
Dysphagia	7	4	3	1	
Excess Saliva	5	2	3	1	
Hoarseness	3	2	1		
Hiatal Hernia	2		1	1	1
Achalasia	2		2		
Tarry Stools	1				1

SUMMARY AND CONCLUSIONS

The esophageal aspects of vagotomy for peptic ulcer have been considered.

A brief review has been given of the anatomy and physiology of the vagus nerve, of the operation and its previously reported effects upon the esophagus, and of the discrepancies between the results of animal experiments and human procedures.

The symptomatology, roentgenologic data and esophagoscopic findings in a series of 31 ulcer patients who had undergone vagotomy have been recorded.

Pyrosis, substernal discomfort, eructation, regurgitation, dysphagia and excessive salivation are not uncommon sequelae of vagotomy and may or may not be accompanied by esophagoscopic findings.

Hiatal hernia, achalasia and esophageal ulcer were observed and may be listed as additional rare complications of the operation.

The esophageal effects of vagotomy, as the operation is now performed, are of relatively minor significance and offer no contraindication to the procedure.

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DISCUSSION

DR. KENNETH A. PHELPS (Minneapolis, Minn.): I have had the opportunity to help one of our local surgeons in his follow-up of a series of cases in all of which he did the vagotomy through the chest. His group comprises 50 cases of about two or more years' duration, and 50 in less than 2 years. Postoperative symptoms of esophageal obstruction were found in about 10% of the patients.

In two of these patients there was a history of complete obstruction of the esophagus following ingestion of food. A piece of meat became lodged in one patient and chemical dissolution was successful; in the other case it cleared up spontaneously.

Patients who have symptoms of cardiospasm were directed to drink hot or warm water in the morning before eating. None of them, since they have been doing that, have had much difficulty. In most of these patients the symptoms have cleared up spontaneously.

It seems that postoperative esophageal symptoms are of minor importance on deciding on this type of surgery

DR. STANTON A. FRIEDBERG (closing): The points brought out by Dr. Phelps confirm the experience of the essayist and of others. The temporary sensation of esophageal obstruction which he mentioned seems to occur in from 10 to 25 per cent of patients. It has often been observed that if these patients swallow liquids just before attempting to swallow food, they will be relieved of their obstructive symptoms. In all of the patients in the present series the symptom of dysphagia eventually resolved spontaneously and dilatation has not been found necessary. This of course speaks for some localized trauma to the esophagus or some temporary dysfunction as the responsible factors; certainly no permanent disturbance of the esophageal nerve supply is engendered as a result of vagotomy as it is now performed. Thank you very much.

LXXIV

UNUSUAL FOREIGN BODY IN A SECONDARY BRONCHUS

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Several years ago Seydell[1] reported a case of spontaneous perforation of the chest wall by an aspirated foreign body. He recorded the fact that the beards of various grains such as barley, wheat and oats, or grasses were able to propel themselves and travel through the bronchial tree, even to the point of penetrating the chest wall. In fact, in each of the eleven cases included in Seydell's report, the foreign body passed through the bronchopulmonary tissues and the pleural cavity to make its appearance in the chest wall.

This report refers to a case in which a head or beard of grass did not go all the way, but traveled only as far as a secondary bronchus, to create an extremely difficult diagnostic problem.

Case report—A male adult, twenty-seven years of age was admitted to the Birmingham Veterans Hospital at Van Nuys, California, September thirteenth of last year. He stated that three and one half years before, he had pneumonia with fever, cough, hemoptysis and nausea which lasted two weeks. Since that time he had experienced episodes of cough and expectoration of blood every three to six months. Two weeks before admission he experienced sharp pain in the left lower chest. This was aggravated by deep breathing; his temperature rose to 104 degrees. Following this he experienced paroxysms of coughing which at first were short and sharp in nature. Subsequently he expectorated yellowish or greenish-yellow sputum which was tinged with blood. This was climaxed by a severe and fairly copious hemorrhage from the lung.

Prior to his admission to the hospital the patient had been under the observation of a well-known chest physician. He had had the benefit of heavy doses of sulfonamides and antibiotics. Bronchograms taken about a year before admission were interpreted as indicating bronchiectasis in the left lower lobe.

Reviewed in the Veterans Administration and published with the approval of the Chief Medical Director. The statements and conclusions published by the authors are the result of their own study and do not necessarily reflect the opinion or policy of the Veterans Administration.

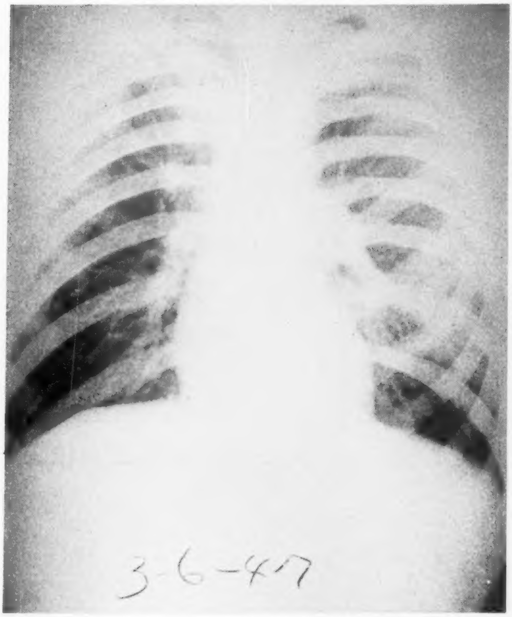

Fig. 1.—Antero-posterior view of chest showing shadow in left lower lobe.

The patient's past history and review of systems were essentially negative.

Physical examination revealed a well-developed and well-nourished white male adult, not acutely ill. The chest was symmetrical and clear to percussion. During auscultation diminished breath sounds were heard at the left base, posteriorly. There were a few moist, scattered inspiratory rales in the same area. The heart was normal. The abdomen and the remainder of the physical examination were also normal.

The initial impression was bronchiectasis of the left lower lobe.

On admission the hemogram was within normal limits; the urinalysis was negative; serology was also negative. Culture of the bronchial secretions revealed a heavy growth of alpha streptococcus and a moderate growth of non-hemolytic staphylococcus albus. Repeated hemograms were within normal limits.

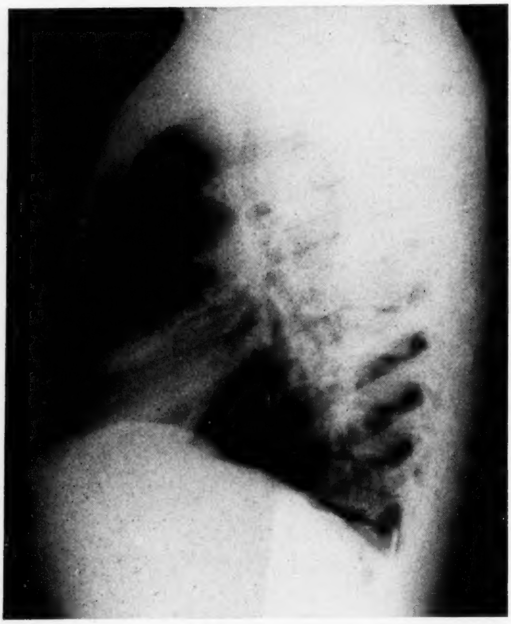

Fig. 2.—Lateral view of chest showing shadow in left lower lung field.

X-ray of the chest on admission showed marked infiltration and opacity of the lower left lobe of the lung; there was some accentuation of bronchial markings extending to that area. The horizontal fissure was within normal limits. The trachea was in the midline; the heart and aortic shadows were normal.

Repeated bronchograms failed to disclose the presence of sacculation or dilatation of the bronchi of the left lower lobe or the lingula. There was no evidence of bronchiectasis. Bronchoscopy was performed several times. At bronchoscopy the only findings were hyperemia of the left lower lobe bronchus with some purulent exudate.

During his period of observation and study the patient was given heavy doses of antibiotics, none of which influenced the x-ray shadow of the left lower lobe.

Planigrams at the twelve centimeter level revealed a dense nodular infiltration of the left lower lobe without honeycombing or

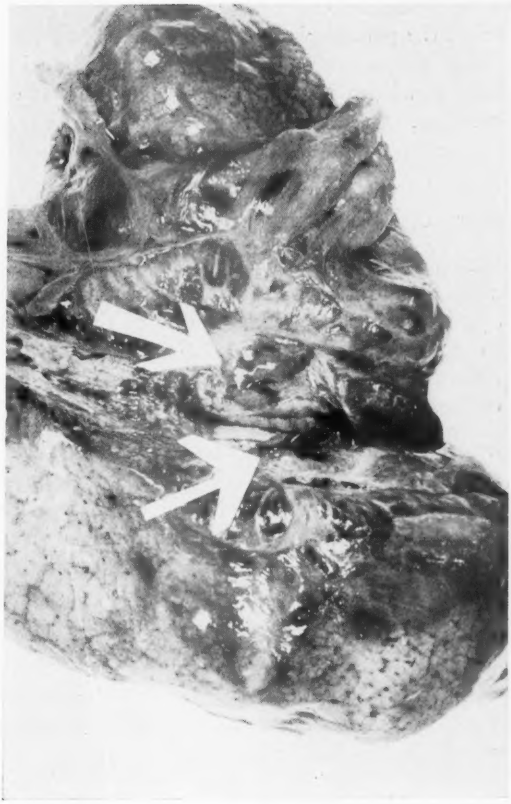

Fig. 3.—Surgical specimen showing grass head in secondary bronchus.

evidence of cavitation. During his stay in the hospital the patient experienced no gross hemoptysis.

A little more than a month after admission the patient was brought before the Surgical Therapeutic Board and was accepted for surgical exploration. On November second a lobectomy was performed by Dr. Joseph Weinberg. On section of the excised lobe it was found that the pathological process was due to a foreign body granuloma. The foreign body was a grass head or beard which had been accidentally inhaled. The postoperative course was uneventful. The patient was discharged well nineteen days after operation. He did not remember having inhaled a foreign body.

A review of the planigrams, after surgery had been performed, disclosed that one of them showed the foreign body.

The pathologists report was as follows: "The specimen consists of the entire left lower lobe, containing a white area six centimeters in its greatest diameter. To one side of this is a twig which measures one centimeter in length. This is apparently the center of the zone of white tissue. There is apparently no communication with the bronchus."

Microscopically, the sections showed areas of marked proliferation of fibrous tissue which extended into the parenchyma of the lung. The adjacent alveoli were markedly compressed and atelectatic; the alveolar walls were thickened. Many of the alveoli showed epithelization. In the alveoli many histiocytes containing lipoid material were seen. Numerous lymphocytes and plasma cells were present. The pathologic diagnosis was: pulmonary atelectasis and fibrosis.

A case of unsuspected foreign body which could not be detected by repeated x-ray studies, bronchoscopies, bronchograms, or planigrams was diagnosed and brought to a successful conclusion as a result of surgical exploration. Such would not have been the case had it not been for the excellent team work and coordination of the various departments interested in this type of case.

It reflects the great progress which has been made in the diagnosis and treatment of chest conditions in recent years.

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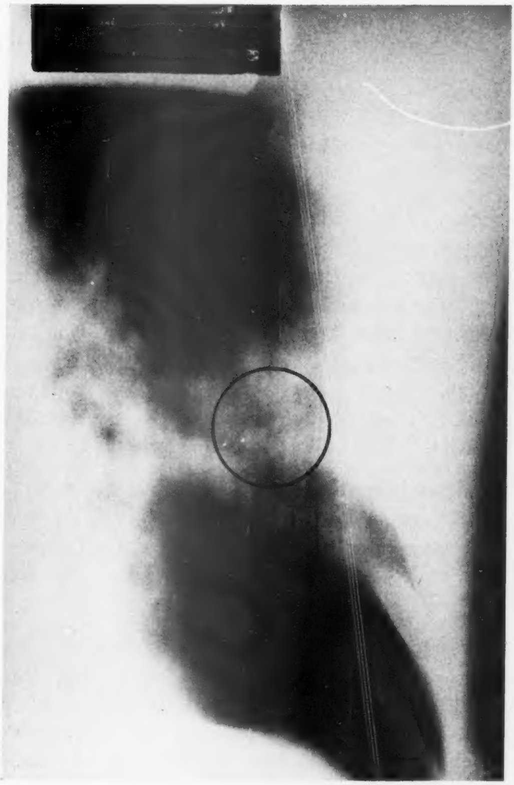

Fig. 4.—Planigram showing shadow in left lower lobe in the center of which can be seen another shadow indicating the foreign body. This was noted after surgery.

LXXV

CARDIOSPASM OR ACHALASIA OF THE CARDIA

SOME PERSONAL OBSERVATIONS AND PRACTICAL CONSIDERATIONS

WITH A PRESENTATION OF 7 CASES OF CARDIECTOMY

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Cardiospasm or achalasia of the esophagus may be defined as an abnormal state of the terminal esophagus characterized by prolonged closure of the lumen of the esophagus at its cardiac end without obvious or apparent gross organic stenosis, which prevents the normal passage of food and drink from the esophagus into the stomach. This obstruction to the passage of the esophageal contents into the stomach may result in dilation of the esophagus or hypertrophy of the walls of the esophagus or both hypertrophy and dilation above the closed area at the cardia.

This condition is manifested clinically by dysphagia and regurgitation of the esophageal content, including food, fluids and salivary secretions. It causes discomfort, even severe pain, in the upper abdomen and chest, and the pain may be referred into the neck, shoulder, arm and back.

Much has been written about cardiospasm or achalasia under many titles. Some of the more common are: congenital, idiopathic and simple dilation of the esophagus, preventriculosis, diffuse dilation of the esophagus without anatomic stenosis, esophageal spasm, hiatus esophagismus, phrenospasm, achalasia of the cardia, nervous affections of the esophagus, and fibrosis of the terminal esophagus. In this paper the term "cardiospasm" is used loosely to include all the above conditions irrespective of the supposed etiology.

ETIOLOGY

The true etiologic factors which produce cardiospasm are not understood. Cardiospasm has been found to be associated with numerous conditions. Various theories have been advanced to explain its production but there has been no general agreement by

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Read before the meeting of the American Broncho-Esophagological Association, San Francisco, May, 1950.

observers who have investigated it. Among the possible causes of cardiospasm may be listed mechanical obstruction such as kinking, twisting, angulation and elongation of the esophagus owing to abnormal states of the esophagus itself; external mechanical factors as pressure about the lower end of the esophagus from the heart, aorta, diaphragmatic crura, spasm of the diaphragmatic muscles, pressure from the tips of the lungs, and from the liver; and periesophageal and intraesophageal inflammatory changes with resulting fibrosis in and about the lower end of the esophagus. A second hypothesis is that there are neurogenic, neuromuscular or reflex phenomena which include degenerative changes in the vagus nerve with paralysis of the circular musculature of the esophagus, primary spasm of the lower end of the esophagus, reflex closure or spasm of the lower end of the esophagus from acid secretion in the stomach, from peptic ulcer or other gastric lesions, from gallbladder disease and from lesions or abnormalities of other abdominal organs. The destruction of the neuromuscular plexus of Auerbach from deficiency states, as a lack of vitamin B, from toxic states and poisons and from esophagitis or infection within the esophageal wall, psychic states or psychosomatic influences are considered by some to be ample etiologic factors.

Vinson[1] is of the opinion that cardiospasm is produced by a disturbance in the nerve muscle mechanism of the esophagus and cardia, probably from degenerative changes in the vagal nerve supply, resulting in overaction of the sympathetic fibers.

Mosher[2] stated that the essential lesion is a tubular narrowing of the terminal portion of the esophagus in the crural canal and especially in the crural ring. This is the result of fibrosis of the periesophageal connective tissue and of the musculature of the esophagus and is caused by infection. He prefers to call this condition fibrosis of the terminal portion of the esophagus rather than cardiospasm.

In the normal individual the lower portion of the esophagus remains closed except at the time of passage of the esophageal content into the stomach. Most observers believe that there is not a true anatomical sphincteric muscle in this area. A few cases have been demonstrated, however, in which there is hypertrophy of the muscles about the upper end of the stomach and the lower end of the esophagus to such an extent that it may be called a true sphincteric muscle. It seems most natural that, when observers cannot even agree on the presence or absence of a sphincter at the lower end of the esophagus, there might also be disagreement on the effect of the nervous mechanism regulating this area. Some observers find spasm, others find no spasm, only the lack of opening, and others find mechanical and fibrotic changes to account for the condition. Perhaps all are correct in certain cases and I believe it is true that a combination of the above factors may be present.

In the majority of cases cardiospasm became manifest clinically in the third and fourth decades of life, but it has been reported in considerable numbers in infancy and childhood, and may develop in old age.

The sex ratio has been reported by Vinson to be 3 males to 2 females. My experience is that the cases have been about equally divided between men and women. There seems to be no hereditary tendency. A few cases have followed trauma, others have been known to follow injury or disease of the brain stem or vagus nerve, and it also may follow infection and toxic states. Cardiospasm has been associated with gallbladder disease and gastric and duodenal ulcer frequently enough so that these conditions should be looked for in patients who have cardiospasm. Psychic trauma, nervous tension and intense worry are believed by some to be true etiologic factors; certainly such conditions do accentuate the symptoms of cardiospasm, but many writers believe they are of little importance. There seems to be no one particular disease or condition which is prone to develop or to be associated with cardiospasm. In a large proportion of the cases of cardiospasm, the onset is insidious and no definite factor can be found to account for, or explain, its origin.

PATHOLOGY

The gross pathology as described from autopsy material has been well summarized in the Proceedings of the Royal Society of Medicine, Section of Laryngology,[3] 1918-1919.

The gross description of the esophagus in cardiospasm varies markedly. It is greatly influenced by the duration of the disease, the completeness of the obstruction, the infection present on the mucosa and in the esophageal walls, and by the extent of dilation of the esophagus and the hypertrophy of the esophageal musculature.

Dilation is the most striking characteristic of the postmortem specimen, and usually extends throughout the length, from the cricopharyngeus to the diaphragm, or even through the diaphragm. In the less advanced cases the esophagus is usually spindle shaped. In the more advanced cases, however, the dilation may be irregularly shaped, and the irregularities may even be haustral-like or resemble diverticula associated with the generalized enlargement of the esophagus. Usually, the greatest dilation is in the lower and middle thirds of the esophagus.

The outer longitudinal coat of the esophageal wall is usually not greatly changed in thickness, the principal thickening being in the marked hypertrophy of the circular muscular coat and in the infiltration of fibrous tissue between the muscular coat and within the muscular coats themselves. The thickness of the walls varies greatly in different areas in the same case and also in different cases. A striking feature is that most specimens show no more marked increase in the musculature of the circular coat of the cardiac end of the esophagus than is found elsewhere in the esophagus. An occasional case will reveal marked muscular hypertrophy of the circular musculature without dilation or with only minimal dilation of the esophagus.

The mucosal lining of these esophagi also varies greatly. This mucosal lining can be visualized in life as well as in the postmortem specimen. The mucosa varies from normal or a slight change in the mucosa to greatly thickened, white, epithelial plaques or leukoplakia, with pebbly fibrous nodules. Often there may be some papillomatous change and in other areas there may be patches of desquamation and true ulceration with complete loss of the mucous membrane. In 3 patients with long histories of cardiospasm (that is, in less than 1 per cent), carcinoma had developed within the chronically inflamed and ulcerated mucous membrane.

Microscopic study of sections of the esophageal wall from various areas of the greatly dilated esophagus has been carried out by Mosher and McGregor.[4] They reported an increase of connective tissue and round cell infiltration, and they noted that blood vessels were increased in number and size; both arteries and veins shared alike in the enlargement. They found Auerbach's plexus most abundantly at the lower portion of the esophagus where the involuntary muscular fibers predominate, but they also found ganglion cells in smaller numbers at the upper end of the esophagus among the voluntary muscle fibers. In the terminal portion of the esophagus no ganglion cells were found in 100 sections made. Fibrosis of the ganglions was not found. In another paper Mosher[2] described the microscopic findings in cases of cardiospasm and laid particular emphasis upon the fibrous tissue changes in the esophagus. These changes were greatest in the tubular, constricted lower portion and the circular layer was almost completely replaced by fibrous tissue. Fibrous tissue changes also occurred in the neighboring diaphragm and diaphragmatic crura. In these particular cases Auerbach's plexus showed no involvement except that there was some fibrosis of the plexus at the point where there were connective tissue changes in the esophagus, that is, at the terminal portion. The esophagi of patients who had cardiospasm all showed chronic infection, and in 2 cases there was extensive periesophageal infection.

SYMPTOMATOLOGY

The cardinal symptoms of cardiospasm are dysphagia, regurgitation and pain. Dysphagia is usually the initial symptom. In most cases the onset is insidious, often intermittent and more noticeable with certain foods. This symptom may gradually increase so that dysphagia is constant with all foods and all liquids taken. The dysphagia, however, may be of sudden onset, and severe and constant from the beginning. In many cases there is a history of almost complete or even complete esophageal obstruction for periods of from a few hours to several days. Usually warm fluids are tolerated better than cold and often fluid is taken to force food into the stomach. Many patients find it advisable to avoid cold liquids, ice cream, raw fruits and vegetables.

In the course of time the esophagus may become greatly dilated and hold as much as 60 ounces. Many patients learn to avoid various foods which give them marked difficulty and also learn to assume a position which helps them to force food and fluids through the esophagus into the stomach, and many use an abundance of liquids for this purpose.

Regurgitation of food occurs to some extent in practically all cases of cardiospasm. It may occur immediately after ingestion in the early cases, before the esophagus dilates to any great extent. The amounts regurgitated may be small and occur infrequently. On the other hand, there is often a period in which practically all the food ingested is returned. When cardiospasm has existed for years, the esophagus dilates to such size that considerable fluid may be taken without regurgitation. In such cases the food may settle in the lower end of the esophagus and when the patient drinks fluids and swallows large amounts of salivary secretion, only the supernatant fluid and salivary secretions may be regurgitated, with very little food content. Again, it is not uncommon for patients to regurgitate food that had been taken several days previously.

Dysphagia and regurgitation go hand in hand and this combination makes eating in public embarrassing for the afflicted person; occasionally he cannot eat in the presence of other members of his family. A patient may start a meal, excuse himself from the table, regurgitate at least a portion of that which has been taken, and return to the table to continue the meal. Regurgitation takes place less easily in the upright position. When the patient is in the prone position, especially when the muscles are relaxed during sleep, regurgitation frequently takes place and the fluid and food particles so regurgitated are often aspirated, causing laryngeal spasm, a sense of strangulation or suffocation, and marked interference with sleep. This may lead to infection of the upper respiratory tract and pulmonary disease, including laryngitis, tracheitis, bronchitis, bronchiectasis, pneumonitis with pulmonary fibrosis, pneumonia and lung abscess.

Practically all patients with cardiospasm complain of pain or discomfort, that is, a sense of pressure or of blockage or obstruction, between the throat and upper abdomen. The pain may be mild and of short duration, being present when food is taken, or it may be persistent. It varies from this mild type to a very severe pain similar to that experienced by patients with coronary heart disease. The pain may be referred to the precordial area and to the neck, shoulder and even into the arm. Occasionally the more severe types may be mistaken for gallbladder colic and the pain extend through to the back. The pain is unpredictable and may come without apparent reason; however, it is more often associated with the ingestion of certain foods. It is likely to follow any manipulation of the esophagus and may occur without provocation during sleep. Pain, as a rule, is more acute and pronounced in the early course of the disease and is less pronounced in old established cases with marked dilation; yet patients with established disease may continue to have severe pain, sometimes with remissions and exacerbations.

SECONDARY SYMPTOMS

Hiccup occurs in many cases of cardiospasm, but as a rule is not severe. Belching takes place, or at least air is swallowed in an attempt to force food into the stomach, and often this is returned.

Constipation is a common association of cardiospasm and results from improper diet and limited food intake. Loss of weight and strength from starvation occurs in a number of patients; however, it is surprising how well nutrition and a fair general health is maintained in others. Cough and sputum from chronic infections in the tracheobronchial tree and lung secondary to aspiration do occur. When the esophagus is very large, dyspnea may occur from pressure on and displacement of the lung, and a mistaken diagnosis of asthma has been made. Hemorrhage and recurrent chills and fever occasionally are present secondary to ulceration and infection of the esophageal wall.

DIAGNOSIS

In many cases a long history and typical story of dysphagia, regurgitation and pain make a diagnosis of cardiospasm almost certain. On physical examination one fails to elicit the normal swallowing sound of food and air passing from the esophagus into the stomach. Fluoroscopic examination of the esophagus with the barium mixture should be carried out in all cases. When the esophagus is greatly dilated and contains much food or debris, it should be emptied by lavage. This is carried out with a large stomach tube and an aspirating bulb such as is used for gastric lavage. Mediastinal broadening and roentgenologic shadows suggesting tumor of the mediastinum may be caused by the enlarged esophagus.

The characteristic finding with the fluoroscope is a smooth, pointed obstruction at the cardia with moderate or marked dilation of the esophagus above the diaphragm. The esophagus may not empty until the column of barium is 6 to 8 inches above the cardia, and a column of this height may remain for a long period of time after the barium is given. In the more severe cases, the esophagus may not empty. It is important to note filling defects; they may be the result of retained food, but if the esophagus is properly cleansed, a filling defect may represent a new growth. Carcinoma occasionally develops in the esophageal wall, and especially in patients with longstanding disease. It seems peculiar that it does not occur more often.

Esophagoscopy is of real value in the diagnosis of this condition. If the esophagus is large or has retained secretions, esophagoscopy should be preceded by esophageal lavage. On passing the esophagoscope a lens is placed in the proximal end of the esophagoscope and the esophagus is inflated, distending it so that its walls can be observed in all portions. The distention of the esophagus with air also permits one to find the dimple or pucker readily, indicating the closed lower passage through the crural canal. Gentle pressure of the instrument usually permits the passage of the esophagoscope to the stomach. Seldom does the air pressure so used open the lower end of the esophagus in cases of cardiospasm. After the esophagoscope has passed, however, the air pressure may be enough to hold the lower end of the esophagus open. During the initial esophageal examination, a Tucker-Plummer dilator may be passed through the esophagoscope and expanded, dilating the closed area. Following the dilation, the inflation of the esophagus and stomach with air frequently will be sufficient to hold the cardia widely open. In some instances slight bleeding will result from this dilation.

Patients who give a short history of esophageal obstruction should have an esophagoscopy. Carcinoma of the stomach involving the lower end of the esophagus has been found by esophagoscopy in a number of patients even though a definite history or radiologic evidence was not obtained. Vinson[1] has stated that the passage of an esophageal sound is more important than is esophagoscopy in the diagnosis of cardiospasm, and that a number 60 French sound can be passed into the stomach without more than slight elastic resistance at the cardia. Vinson passes this instrument over a previously swallowed and well-anchored thread. If there is difficulty in passing a large olive-type bougie it is probable that a stricture or new growth exists.

The differential diagnosis of cardiospasm is more difficult in the early stages than in the later stages. If pain is a presenting symptom, the condition may be confused with cardiac conditions and disease of the gallbladder. Stricture of the lower end of the esophagus and especially that due to carcinoma beginning in the upper portion of the stomach or lower end of the esophagus can be and frequently has been confused with cardiospasm. With complete fluoroscopic, roentgenologic and esophagoscopic studies, however, the possibility of cancer can be well evaluated. Benign stricture, diverticulum and ulcer of the lower end of the esophagus and diaphragmatic hernia sometimes produce symptoms similar to, and at times can be present together with cardiospasm.

MEDICAL TREATMENT

One must not lose sight of the fact that in cardiospasm the passage of food from the lower end of the esophagus into the stomach is not normal. To obtain relief, this obstruction must be overcome. Rarely if ever will medical treatment alone accomplish this end. Certainly, if medical or neuropsychiatric therapy is to be employed in these cases, it should be only in the early cases without marked anatomical changes. In view of the positive successes obtained by dilation, medical treatment as a means of relieving the obstruction rarely seems justified.

Treatment of Secondary Conditions. Medical care is indicated to relieve the secondary conditions resulting from the esophageal obstruction and infection, malnutrition or starvation. In such cases, supportive treatment by the parenteral use of fluids, vitamins, glucose and amino acids is indicated until such time as feeding can be resumed by way of the gastrointestinal tract. Also, the infected and diseased esophagus may require cleansing by proper lavage and the infection may be combated by the employment of sulfonamide compounds and antibiotics. It may be necessary to teach a patient to use a tube for esophageal lavage or to empty the esophagus, especially before retiring, by drinking warm fluids, assuming a position which turns the esophagus upside down, thus aiding in regurgitation of fluid. In addition, patients who are disturbed by regurgitation during their sleep should sleep with the head and shoulders well elevated so that gravity will help to maintain the fluid content in the lower end of the esophagus.

Dietary management is, of course, not important in cases in which complete relief is obtained immediately from dilation. For those who are not relieved by dilation dietary management is of help. The food should be well chewed or if the patient is unable to chew well, because of lack of teeth or for some other reason, the food should be properly prepared by passing it through a grinder, ricer or sieve so that it is finely divided. Most foods should be cooked and raw fruits, vegetables and tough meats should be avoided. Even the coarse fiber of cooked fruits and vegetables should be eliminated. It may be well to precede a meal by a warm drink which may have some beneficial relaxing influence on the lower end of the esophagus. Warm fluids following meals may help discharge the esophageal content into the stomach and wash it free of food debris.

Patients under nervous tension or those who have psychic trauma may benefit from sedatives or psychiatric care, and others may be helped by high vitamin intake.

Drugs for the Relief of Cardiospasm. Various antispasmodic drugs have been tried and some have been found helpful. Atropine and belladonna have been of little value. Vinson cited a case, however, in which the patient was treated by Plummer[5] and marked relief was obtained following hypodermic administration of 1/4 grain of atropine.

Aminophylline was found to be especially useful by Browne and McHardy.[6] The nitrates have been known to relax smooth muscle. Krantz, Carr and Forman[7] described the pharmacology of octyl nitrite. They found it to be less toxic and its effects longer lasting than amyl nitrite, and suitable for use with an inhaler. Ritvo and McDonald[8] have reported on the administration of amyl nitrite and nitroglycerine, which temporarily relieved the stenosis of the esophagus in many cases of cardiospasm, permitting the passage of fluid and food into the stomach. The effect of amyl nitrite is of short duration. The side effects may be marked and the odor objectionable to many patients. Giddiness and fainting, with loss of consciousness, may occur. This drug is best used when the patient is recumbent. Nitroglycerine also produced untoward reactions, but they were milder than those produced by amyl nitrite. Flushing and perspiration were the most common reactions. Nausea and vomiting were likely to occur. Throbbing and pounding headache with giddiness and vertigo was present in several patients. Krantz, Carr and Forman reported that there was a striking difference in the tolerance of various individuals and the drugs proved disappointing for other than temporary relief. In some cases nitrates produced relaxation, with filling of the stomach.

Field[9] has reported the use of octyl nitrite in patients with achalasia of the cardia. He found octyl nitrite, used in an inhaler, to be effective and practical for obtaining symptomatic relief of achalasia in 4 cases. The increase in the feeling of well-being was striking. No toxic symptoms were observed. The effectiveness of the octyl nitrite inhaler varies from seven to twenty-eight days. Octyl nitrite was found to be quicker in action than nitroglycerine and produced less nausea than did amyl nitrite. The patients continued to respond well to octyl nitrite after prolonged usage. A case was reported, however, in which tolerance was obtained to this drug. Field believed that the supposition of tolerance was wrong because the inhalations were given five minutes before a meal. His practice is to give the inhalations after a meal has begun or when distress is evident. He stated that the action of the octyl nitrite on the cardia is transient and rarely lasts more than five minutes, so that an inhalation five minutes before the meal is not likely to have much effect. He also pointed out that after continued use of the drug its action may have become more rapid and of shorter duration. This effect was noted in his previously reported cases. He further reported that a year and a half later, from November 30, 1944, to May 1946, tolerance did not develop in his 4 patients. They no longer had side effects or any toxic effect. The parotid gland became enlarged and lobular collapse of the lung developed in 2 patients. He has given steam inhalations to his patients who required octyl nitrite inhalations.

Fleminger and Smith[10] reported a case of achalasia following injury to the head. A psychic element was thought to be responsible and the intravenous use of sodium amytal and psychic suggestion were unsuccessful, but one inhalation of octyl nitrite was followed by relief.

Etzel[11] has described cases of megalo-esophagus or cardiospasm, megacolon and mega-ureter which he believed may be caused by chronic vitamin B deficiency. Stinson[12] has reported on the use of vitamin B and thiamine chloride in the treatment of cardiospasm. This vitamin was given intramuscularly and by mouth to relieve the symptoms of cardiospasm. The dosage varied from 3000 to 6000 units and was given daily or at intervals of from three to four days by intramuscular injections.

THE TREATMENT OF CARDIOSPASM BY DILATION

Three types of dilators are in common use in the treatment of cardiospasm by dilation of the cardiac canal or crural canal. They are the olive type of bougie on a flexible staff, the expansible dilator and the weighted, shot or mercury-filled bougie. These instruments may be combined into one and used in the treatment of cardiospasm by dilation.

The olive-tipped bougie consists of the olive shaped dilator which may be as large as 2 cm in diameter or 60 French. It is mounted on a flexible shaft receded by a flexible finder which is passed over a previously swallowed thread. In passing the instrument, the thread is drawn taut and guides the flexible finder through the lumen of the esophagus, preventing perfortion of the esophageal wall. This type of instrument has been advocated by Plummer and Vinson and is also used for the dilation of benign and malignant strictures of the esophagus, but the largest size usually employed for this purpose is a 45 French.

Vinson, who advocates and uses this procedure, stated that 10 per cent of the patients are relieved to a marked extent and half of this 10 per cent are completely and permanently relieved; the remaining patients require further dilations with sounds or expanding dilators. Vinson further stated that prior to 1925 he employed the hydrostatic dilation of the cardia in all of his patients. An occasional fatality from rupture of the esophagus as a result of overdistention led to preliminary dilation with a 60 French sound. Since this procedure, hydrostatic dilation has been used in over 450 cases without a fatality. When symptoms recur in a few months the sound should be again passed if the dilation of the esophagus above the obstruction is not marked; the risk of hydrostatic dilation, however slight, need not be assumed.

The Dispensable Rubber Bag Dilator. Sixty years ago, Russell[13] first used a dispensable rubber bag dilator, and in 1898 published a paper containing the following statements: "As to treatment that in my hands has given good results consists in stretching the stricture by an expanding dilator to a caliber approximating that of a normal esophagus at that point. This is not done suddenly or at one operation but with bags of increasing size at several sittings.

"When bougies failed, I accordingly made an instrument consisting of a sausage shaped silk bag rendered air tight by a very thin rubber bag within mounted on the end of a tube or hollow bougie. This was passed through the stricture in the collapsed state and then blown up by an air syringe when in position."

Fundamentally, this is the procedure used today. There have been many modifications, only a few of which are mentioned. In this country Plummer's modification of this instrument popularized its use. Plummer used water or hydrostatic pressure for the distention of the bag and measured the pressure on the gauge in feet of water, using up to 22 to 24 feet or a pressure of 9 to 10 pounds per square inch.

Vinson has continued to use this dilator and passes it over a previously swallowed thread (quoting from Vinson) "until the center of the distal third of the dilator rests in the cardia. The dilator is held in place and distended with water until the gauge indicates a pressure of 22 to 24 feet of water. The patient usually experiences discomfort as the cardia is dilated unless the dilator slips out of place into the stomach or esophagus. The usual displacement is into the stomach. When difficulty is experienced in maintaining the position of the dilator in the cardia it may be necessary to introduce the instrument into the stomach, distend the bag with the water and pull it forcibly against the cardia. By releasing a small amount of water, entrance of the instrument into the esophagus can be felt. When a portion of the bag has been pulled into the esophagus, the water pressure is renewed rapidly and the cardia usually is effectively distended . . . distending the dilator with water is preferable to air because it is more easily maintained with the former; also, dilation is less hazardous because of the lack of compressibility of water—it would seem that fluoroscopy might facilitate more accurate introduction of the dilators into the cardia. But such is not the case. The handicap of attempting dilation in a dark room is greater than the aid which can be obtained by visualizing the dilator in the esophagus."

Mosher has added two very useful modifications: one, the use of a barium striped bag (bag with linear impregnation of barium) which enables him to examine the esophagus visually and to dilate it before the fluoroscope. Secondly, instead of a metal olive tip on the end of the dilator he introduced a flexible finder some 3 to 4 inches long, with a heavy perforated tip about ⅜ inch in diameter. In only an occasional instance will this finder fail to pass into the stomach from the esophagus and in these occasional cases the passing of a finder over a previously swallowed thread is then possible. It is rare to find a patient with such angulation that the Mosher type dilator cannot be passed from the esophagus into the stomach over a previously swallowed thread, yet I have had a few cases in which I was unable to do this with fluoroscopic guidance.

Quoting from Mosher, "The ideal treatment of narrowing of the terminal portion of the esophagus is dilation under fluoroscopic vision. Mild cases of fibrosis respond readily. Even cases of long standing may dilate sufficiently to give a clinical cure. In old cases where the esophagus is much dilated and sags markedly to the right, dilation—always under sight—may be tried cautiously. In such patients the esophagus is generally thin and its musculature degenerated. They are dangerous cases to handle by dilation. These patients are candidates for gastrostomy or for an anastomosis between the lower end of the esophagus and the fundus of the stomach."

Mosher also stated that 3 to 4 pounds pressure is all that is necessary to dilate the crural dent or crease which is a constant finding in cases of cardiospasm. He stated that he once carried the pressure to 6 pounds, at which point the patient fainted from pain. In one case, a pressure of ½ pound overcame the niche of a crural dent. Mosher also equipped the instrument with a luminous dial so that it could easily be read in the fluoroscopic room.

A third modification which I recommend and employ is that by Tucker for use through the esophagoscope. With this instrument, the dilation can be carried out at the time of the initial esophageal examination if conditions warrant. It is small enough to pass through the 9 mm full lumen esophagoscope and is so marked that it can be accurately placed. The esophagoscope is then withdrawn a sufficient distance to clear the cardia and the dilator expanded.

I have found the staffs which are used to introduce the Plummer dilator cumbersome and often difficult to remove when in place or uncomfortable if left in the tube of the dilator. A coil spring is used instead. This is flexible, prevents kinking and can be left in place without discomfort during the dilatation.

Dilation with the Hurst Mercury-Filled Bougie. Hurst[14] constructed bougies of large, flexible rubber tubing with a blunt dilating tip. These tubes vary from 33/64 to 46/64 inch in diameter, that is, 39 to 56 French; they are 31 inches long and each bougie contains 1 pound 5 ounces of mercury. The bougie is passed by the physician or swallowed by the patient. Its own weight is sufficient to carry it through the esophagus and cardia into the stomach. Hurst suggested that fluoroscopic visualization be employed at the first passage of the instrument. Increasing sizes are used and the patient may be trained to pass the bougie himself. At the beginning of treatment it may be passed before each meal. As symptoms are relieved dilation may be required less often, the frequency of using the instrument being left somewhat to the patient's own discretion. When symptoms are completely relieved it is no longer necessary to use a bougie.

This instrument has a marked advantage in that it can be quite safely passed by the patient himself, and it may be used when other methods of treatment have failed to give relief, especially when he lives at a distance where dilations may be difficult to obtain.

This instrument cannot be used in all cases, and especially not in those patients who have a tortuous esophagus. Hurst has recommended that this instrument be left in place for fifteen minutes if possible and that the larger size be used if tolerated by the patient. Good results have been obtained, however, with the smaller-sized dilator.

Browne and McHardy have combined the Hurst mercury bougie with the Plummer dilator, and found it effective.

The following are a few personal experiences which most writers have not emphasized. First, in regard to swallowing the D buttonhole twist silk thread, a 10 yard or 30 foot spool is given to the patient. He is instructed to swallow it slowly, not more than 1 to 2 feet an hour during the day while he is awake. The average person is asked to begin swallowing it two days before the time scheduled for the dilation. When all but about 6 feet have been taken, he is asked to make no effort to swallow more; if there is a tendency for the thread to pull, a little at a time may be swallowed to relieve the tension. The thread is tested for anchorage by pulling upon it well before the time of dilation. Patients who have marked difficulty in swallowing may require more than two days to pass the thread, while those who have little difficulty in swallowing may take it more rapidly. If the patient who has difficulty in swallowing takes the thread rapidly, tangling and knotting are likely to occur, which interfere with using the thread as a guide. After the thread has been used as a guide it is cut off at the mouth. The thread is discharged from the gastrointestinal tract usually in one bowel movement. If it is caught between two movements, it may be necessary to use a scissors to cut it.

Following dilation with the Tucker bag through the esophagoscope, the bag is removed, the crural canal reexamined, and some bleeding from the mucosa has been found in several cases. Also, following the dilation with the distensible bag, the bag is always examined for traces of blood or blood stains. I have found blood on the dilator in a number of cases. This finding strongly supports Mosher's observation of the gluing together of the folds through the crural canal. Certainly, there must be some break in the mucosa to produce bleeding. Most writers fail to make clear the amount of pressure and the size of the dilator used. High pressure in a small bag may be safe, while high pressure or the same pressure in a large bag would surely tear the esophageal wall. If the bag is fully distended under 5 pounds of pressure there is no further dilation when 15 pounds pressure is applied.

If, on the application of 5 to 6 pounds pressure, there is a considerable niche in the crural canal, it is safer to use the Hurst dilator or a smaller pneumatic bag with higher pressures, gradually increasing the size of the bag to obtain dilation of the crural canal. In cases with marked fibrotic change gradual dilation is safer than rapid dilation. The danger of spreading infection through the esophageal wall following dilation is always present. In 4 cases mediastinitis followed dilation but the patients recovered following mediastinal drainage. Even though recovery did take place in these 4 cases, it is a serious condition and requires three to four weeks' hospitalization for the patient's recovery.

Failure to obtain relief of this condition by dilation may be due to the fact that proper distention or overdilation was not carried out. Under my own observation a man was given repeated dilations for a period of three years, without satisfactory relief. However, when the esophagus was completely dilated, relief was obtained without further treatment for a period of ten years, at which time the patient died of other causes. One patient who came to the clinic had been under treatment for eighteen years; following adequate dilation complete relief was obtained within three weeks. Another patient, aged 60, with onset of symptoms thirteen years previously, had been under treatment at intervals during this time. The roentgenogram showed the Mosher bag distended to 6½ pounds pressure and there was still a considerable niche in the crural canal. The question arose, would further pressure produce a cure or would it produce mediastinitis and possible fatality? Further dilations have relieved this patient.

Each patient presents his own problem and must be treated individually. The dilatation may be quick and easy to accomplish, with complete relief of symptoms, or it may, on occasion, produce mediastinitis. On the other hand, it may be slow, difficult and hazardous, with much pain and slight relief to the patient. In such cases surgical treatment should be carried out.

SURGICAL TREATMENT OF CARDIOSPASM

Ochsner and DeBakey[15] have reviewed the literature on this subject and have classified the various types of operative procedures which have been advocated and employed for the treatment of cardiospasm or achalasia. The first classification, that is operations directed at the dilated esophagus, can well be discarded as a procedure for the correction of this condition in most instances. Operations directed at the diaphragm may, likewise, be discarded. The results of operations directed at the nerve supply, while having some sound principles, are not likely to produce satisfactory results in old established cases in which operative procedures seem necessary. That leaves operations directed at the cardia. Such operations have been classified into four groups. The first group is dilation, retrograde and transgastric. I see very little reason for employing this procedure, for certainly it is as dangerous as, and no more likely to produce results than, dilation without operation.

Ochsner and DeBakey reported the plastic surgical handling by cardiomyotomy, using the Heller or modified Heller procedure, and collected 104 cases from the literature. Satisfactory results were obtained in 80, recurrence took place in 14, and 4 patients died. Improvement was observed in the 6 remaining cases.

Thirty-six cases of cardioplasty were collected, in 20 of which the incision was made entirely through the esophageal wall and in 14 an extramucous cardioplasty was done. Only one death occurred and recurrence was reported in 1 case. Sweet[16] preferred this method in selected cases of idiopathic dilatation of the esophagus. Ochsner and DeBakey found 2 reported cases of cardiectomy; one patient died of heart disease following the procedure. They commented that these are the only 2 cases that have been reported, and emphasized the objectionable features of the operation. In their opinion, such a radical procedure is justified only in the presence of a malignant growth. There seems to me to be a few cases in which cardiectomy may be the operation of choice even though a malignant growth is not present.

Ochsner and DeBakey concluded that esophagogastrostomy is the most rational procedure to be used in cases of achalasia or cardiospasm in which an operative procedure is indicated. They have collected 88 cases from the literature, with 5 deaths and only one poor result. All operative procedures except cardiectomy are well described and illustrated in their paper.

The following 7 case reports represent cases in which cardiectomy with removal of some portion of the stomach and esophagus has been carried out for the relief of a benign condition, stricture, fibrosis or cardiospasm.

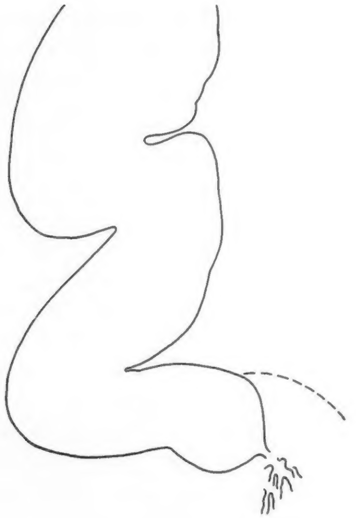

Fig. 1, Case 1.—Outline of barium-filled esophagus which contained 60 ounces. The esophagus is fully distended without evidence of muscular contraction. The outline remained smooth after three weeks of treatment consisting of gastrostomy, dilation and lavage.

REPORT OF CASES

Case 1.—A woman, aged 47 years, was first seen September 15, 1947, with the complaints of difficulty in eating with regurgitation of food for twenty-five years or more and severe attacks of dyspnea for seven years. She had difficulty in taking food because of substernal fullness and the regurgitation of undigested food, resulting in a weight loss of 37 pounds. These difficulties had become progressively worse with coughing, choking sensations and some wheezing. During the past seven years she had had intermittent attacks of severe dyspnea, especially following eating, and associated with coughing and distention of the neck veins. These symptoms were relieved to a considerable extent by regurgitation. She suffered from some dyspnea following each meal and on exercise. She also complained of left frontal headaches, with blurring of vision, and frequent periods of palpitation. Four weeks before coming to the clinic an esophageal examination was made with barium and marked dilation of the esophagus was found.

A general examination showed the patient to be markedly emaciated and chronically ill, weighing 93 pounds. The veins over the right thorax were dilated, and in the supine position there was marked

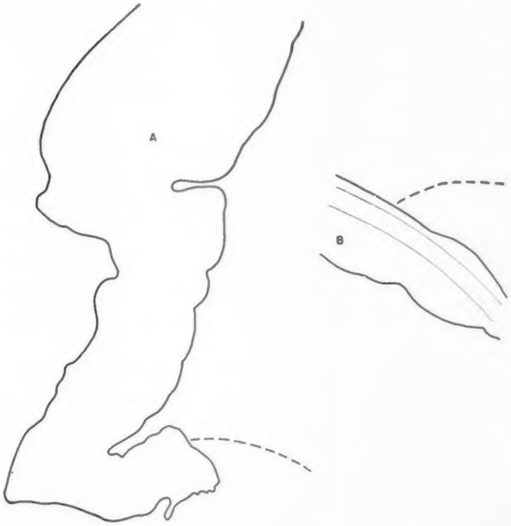

Fig. 2, Case 1.—*A*, Outline of barium-filled esophagus three months after treatment with gastrostomy, dilatation and lavage. The outline of the esophageal wall is now irregular, showing some return of muscular contraction. *B*, Outline of barium striped bag lying in the cardia when distended to 7 pounds pressure.

distention of the neck veins and cyanosis. Marked dullness of the right chest was noted on percussion. Barium studies demonstrated a huge esophagus, as illustrated in Figure 1.

On September 16, 1947, an esophagoscopy was carried out. There were marked inflammation and thickening of the esophageal wall and two very large pouches with a constriction near the hilum. At this time a bronchoscopy was also done which showed deviation of the carina to the right, and the tracheobronchial membranes were red and edematous. Because of the poor condition of the patient a gastrostomy was done for feeding purposes. Further esophagoscopies and dilations were carried out. The esophagus was lavaged daily. Antibiotics were administered. She was discharged home to continue with washings of the esophagus and with her gastrostomy feedings. She returned three months later, having gained 26 pounds. At that time examination of the esophagus revealed marked improvement of the esophageal wall with very little inflammation, but thickening and some leukoplakia were still present. Since there was marked retention in spite of satisfactory dilation of the lower end of the esophagus (Fig. 2 *A* and *B*), the lower 6 inches of the esophagus with the cardia was resected. Following this procedure she has made steady and rapid improvement. No further treatment was necessary. She now eats all foods without difficulty; she works hard and feels quite well. The result is excellent.

Pathologic Report: The little finger could easily be inserted through the orifice but there was organic narrowing of the esophagus. The specimen was 17 cm in length, 12 cm of which was esophagus and 5 cm stomach (Fig. 7*A*). The upper end of the esophageal specimen was 4 cm in diameter and 1.5 cm in diameter at the cardia. The mucosa was edematous with longitudinal folds and regions of yellow opacity. The esophageal wall was 0.7 cm. thick.

The microscopic report was hyperkeratosis and focal ulceration of the esophageal mucosa, hyperplasia, fibrosis and chronic inflammation of the esophageal musculature.

The sections varied in thickness from 1.0 cm to 1.5 cm. The muscle coats, particularly the circular layer, were markedly thickened. The fibrous tissue content was much increased and dense connective tissue surrounded and replaced many muscle fibers in all portions of the wall. This was most marked in the inner portion of the circular coat. There was diffuse lymphocytic infiltration of all layers with intermingled granulocytes. The mucosa was focally thinned and infiltrated by lymphocytes. Nerve fibers were numerous between muscle bands, but no ganglion cells were found. The blood vessels were negative.

Case 2.—A woman, aged 61 years, was first seen on September 13, 1949, with the chief complaint of fatigue. She had been tired for many years, but attributed it to overwork, having raised a family of six children. She also complained of nervousness, difficulty in eating and headaches. She slept poorly and would get up at night, eat a lunch and try to sleep again. Three weeks before coming to the clinic she felt very weak and exhausted. This was followed by the passage of black, tarry stools, and her physician found that she had a severe anemia. A diagnosis was made of intestinal hemorrhage and "cardiospasm." Complete gastrointestinal studies were negative except for the esophagus (Fig. 3), and the source of bleeding was not determined. Transfusions and antianemic therapy were given. On further questioning it was found that her difficulty in swallowing began at the age of 21 years and had continued for forty years. It was much worse at some times than at others; it was always worse with emotional upsets and great fatigue.

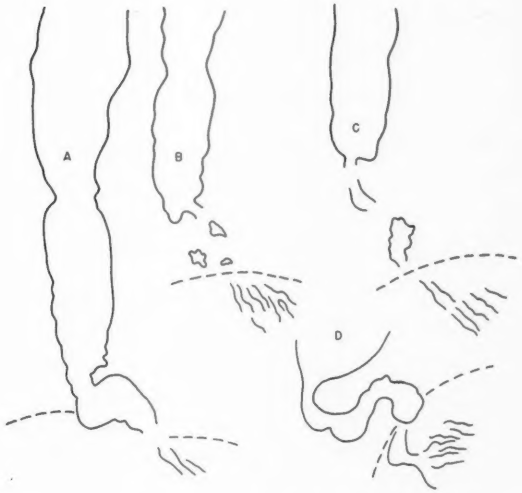

Fig. 3, Case 2.—*A*, Outline of barium-filled esophagus made at intervals, illustrating marked muscular activity near the cardia, which could obliterate the lumen of the esophagus in this area (*B* and *C*) and the tortuosity at the other interval (*D*).

Again, complete gastroenterologic studies failed to reveal the source of bleeding. During her hospital stay an esophagoscopy was carried out. There was no ulceration in the esophagus; it was somewhat tortuous and the lower end of the esophagus was readily dilated with a Tucker-Plummer dilator. Following this procedure inspection revealed slight bleeding from the dilation. Swallowing was considerably improved but not completely relieved. Regurgitation occurred again within two weeks but from then on much less often. Dietary and supportive treatment was carried out by her family physician, and during a four-month period the hemoglobin returned to 94 per cent.

A second severe hemorrhage occurred and the patient returned to the clinic for hospital care on February 17, 1950. At that time her hemoglobin was reduced to 72 per cent. Studies again failed to reveal the source of bleeding. The lower end of the esophagus was visualized and dilated. A definite bleeding area could not be found.

Because the lower end of the esophagus was tortuous and dilations did not give relief, only improvement, of her dysphagia, and because of recurrent severe hemorrhages, operative treatment (cardiectomy) was advised and carried out on March 1, 1950. Marked peri-esophageal adhesions were found at operation. The postoperative course at first was stormy. Dilations were necessary. She was swallowing better than she had in forty years. When discharged she was eating a regular diet; her condition was excellent. The cause of the severe hemorrhage to date has not been explained.

Pathologic Report: The little finger passed through the lumen of the removed specimen (Fig. *7B*) without difficulty but there was some gripping of the finger by the muscular wall. The specimen measured 4.5 cm in length. The gastric mucosa was only 0.7 cm in length and the circular muscle 0.8 cm thick.

The microscopic report was slight chronic esophagitis and fibrosis of the submucosa and muscularis. No ganglion cells were present.

The sections were 0.6 cm in thickness. The circular and longitudinal muscle coats were twice thickened and there was a slight thickening of the muscularis mucosae. There was a moderate increase of fibrous tissue between the muscle layers and diffuse infiltration of lymphocytes in all parts of the wall. The mucosa was thicker than usual, but otherwise negative. Nerve fibers were abundant between the muscle layers but no ganglion cells were found. Blood vessels were negative.

CASE 3.—A man, aged 42 years, was first seen at the clinic July 7, 1947, with a history of difficulty in swallowing both liquids and solids for over a year. He stated that shortly after swallowing almost any food, it was regurgitated as taken. If he ate only small amounts at quite frequent intervals, however, there was less trouble. He felt weak and had lost 45 pounds in weight in the last year. He had continued to work. He was treated by dilation, with slight relief of short duration. Cold liquids were particularly troublesome to him. He did not have pain.

Roentgenograms revealed a dilated, tortuous esophagus and narrowing at the cardia (Fig. 4). On July 10, 1947, esophagoscopy was carried out. The esophagus was considerably dilated; the mucous membrane of the esophagus was somewhat thickened and pale. A narrowed opening in the lower esophagus led to the stomach through which a number 14 Jackson dilator was passed, but the esophagoscope was not passed through this area with ordinary pressure.

Cardiectomy was carried out on July 15. The postoperative course was uneventful and the patient took liquids well on the second postoperative day. He was discharged on the fourteenth postoperative day, at which time he could take meals without difficulty.

On fluoroscopic examination, however, when a small amount of barium was taken, the esophagus became filled. During the filling

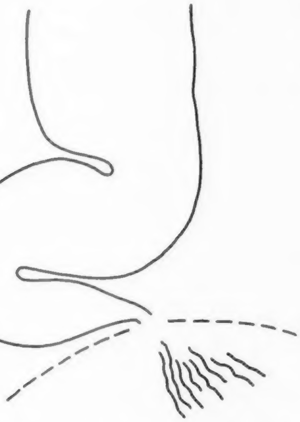

Fig. 4, Case 3.—Outline of barium-filled esophagus, as seen in the roentgenogram.

of the esophagus the barium mixture was retained by the tone of the musculature of the lower portion of the esophagus and did not pass into the stomach. After the esophagus was filled, the esophageal content discharged readily into the stomach. At follow-up examination one year later, June 1948, the patient had maintained his weight but still had some dysphagia. The area of anastomosis was dilated, with further improvement. The result in this case following dilation was good.

Pathologic Report: Following removal of the specimen the little finger could not be passed through the lumen of the esophagus without undue force. After opening, the esophageal segment was 2.3 cm in length and the stomach segment 5 cm in length (Fig. 7C). The esophageal mucosa was not ulcerated but was gathered into six longitudinal folds due to the presence of firm, resilient thickened tissue in the esophageal wall. The lumen at the gastroesophageal junction was considerably narrowed—practically obliterated.

The microscopic report was chronic inflammation of the esophageal mucosa and hypertrophy of the muscularis.

The section was 0.6 cm in thickness. The circular muscle coat was three times thickened. The muscularis mucosae was twice the usual thickness. The longitudinal muscle layer was only slightly thicker than normal. Throughout the muscle coats there was a very slight increase in interstitial collagen. There was scattered infiltration by eosinophils and lymphocytes in the mucosa and all the connective tissue elements of this section. The esophageal mucosa was thin and elsewhere acanthotic. Gastric mucosa was negative. Nerve fibers in the muscle layers were abundant but no ganglion cells were found. The blood vessels and adventitia were negative.

CASE 4.—A man, aged 68 years, came to the clinic April 24, 1947, because of regurgitation of his food during or after meals for the previous three and a half years. Dysphagia and substernal distress had been present for more than twenty years. In December 1943, roentgenograms revealed an esophageal diverticulum. During the three months before coming to the clinic his symptoms had become progressively worse and during this time he had lost 25 pounds in weight. He was weak and cachectic.

Fluoroscopic, roentgenologic and esophagoscopic studies revealed a greatly dilated esophagus with a large diverticulum, approximately 12 cm in diameter, at the lower end of the esophagus. A constriction ("cardiospasm") at the cardiogastric junction was dilated readily. Because of his emaciated condition gastrostomy was performed for feeding. He was discharged May 11, 1947, to return at a later date for further surgical consideration of the esophageal condition. The patient gained markedly in one month's time.

On June 13, 1947, resection of the diverticulum was carried out by Dr. Ralph Adams. No attempt was made at that time to relieve the cardiospasm. Recovery from excision of the hernia was uneventful but the patient was still unable to swallow. On July 3, 1947, a second esophagoscopy was done and Jackson bougies up to size number 16 French could be passed, but larger bougies were firmly gripped by the musculature of the lower esophagus. Because the area of closure was 1½ inches in length, cardiectomy was recommended and carried out on July 8. At that time the gastrostomy was closed and the patient was able to take all food without difficulty. By August 22, he had gained 21 pounds. He ate everything he wished, including steak. Dilations were not necessary. The result in this case is excellent.

Pathologic Report: The little finger could not be passed through the lumen of the esophagus. The opened specimen was 7 cm long, 6 cm of esophagus and 1 cm of stomach (Fig. *7D*). The circumference of the upper portion of the esophagus was 5.5 cm and 3.5 cm at the cardia. The mucosa of the esophagus showed a scar of a previous operation when the diverticulum had been removed. There were numerous sutures on the outer wall of the esophagus opposite the mucosal scar.

The microscopic findings were fibrosis and hypertrophy of the muscle and slight, chronic inflammation.

The section was approximately 1 cm in thickness. The muscularis mucosae and the circular muscle coat were at least two times thicker than normal. The longitudinal muscle layer was not thickened. There was a slight increase in fibrous tissue between the muscle layers and in the submucosa; this was particularly marked in the inner portion of the circular muscle layer and between this layer and the muscularis mucosae. The submucosa and the mucosa were markedly infiltrated by lymphocytes with focal accumulations of these cells about the glands. The mucosa itself was twice its usual thickness and acanthotic. Several glands showed epidermidization of their ducts. No ganglion cells were found, although nerve fibers were abundant between muscle coats. There was increased vascularity between muscle layers and adventitia, more marked beneath the muscularis mucosae.

CASE 5.—A man, aged 69 years, was first seen July 26, 1948, with the chief complaint of dysphagia and distress of twenty years' duration. His symptoms had been very distressing for the past year, and during the past five weeks before coming to the clinic he had very marked distress with pain and hiccup almost constantly. He was relieved somewhat by taking soda and by belching. There was very little regurgitation. He had lost considerable weight during the past year.

The general examination, other than showing weight loss and a foul breath, was not unusual for a man of his age. His blood pressure was only 126 mm systolic and 80 mm diastolic. The fluoroscopic and roentgenologic studies of the esophagus revealed a large diverticulum of the lower third of the esophagus on the right side, with a moderately dilated esophagus.

Esophagoscopic examination demonstrated a large diverticulum containing many food fragments, a dilated lower esophagus and a smooth esophagogastric junction. The stricture was easily dilated. It was thought to be typical of "cardiospasm." A diagnosis was made of fibrosis of the lower end of the esophagus, "cardiospasm," with a large pulsion diverticulum of the lower third of the esophagus. Excision of the lower portion of the esophagus including the diverticulum and the cardiac end of the stomach was recommended, and this was carried out by Dr. H. D. Adams.

The postoperative examination with the fluoroscope showed the fundus of the stomach to have been pulled upward and anastomosed to the esophagus. The anastomosis was adequate and the barium passed through the area without delay. The patient was discharged from the hospital on his twelfth postoperative day.

Follow-up: In a recent letter the patient stated that he still has some of his symptoms, that is some fullness and difficulty in swallowing. He has been requested to come in for observation and possible dilation, but his symptoms have not been severe enough to make him seriously consider this.

Pathologic Report: The specimen (Fig. 7*E*) consisted of a 6 cm segment of esophagus with gastric mucosa at its lower end. Attached to the lower end was a smooth-walled, collapsed sac 6 cm in diameter. The specimen would not admit the small finger through the esophagogastric junction without pressure. The opened specimen showed a smooth mucosal lining.

Microscopic examination demonstrated chronic inflammation.

Complete study was not made.

CASE 6.—A woman, aged 43 years, first came to the clinic September 21, 1946. Her chief complaints were stomach pain, nausea and regurgitation of two years' duration. She had had some regurgitation and a known "cardiospasm" for ten years, during which time esophagoscopy has been performed and dilations carried out, without relief. She stated that she was often made worse by such treatment. She had recently lost 10 pounds in weight. Raw fruits and vegetables were particularly troublesome. At the onset of her trouble there was gradual increase of symptoms, but at the time of entry she experienced nausea, regurgitation and stomach cramps with every meal.

Fluoroscopy and roentgen examination were consistent with benign obstruction of the esophagus in the crural canal with only moderate dilation and marked muscular contractions (Fig. 5). Esophagoscopic examination was carried out on September 27, 1946, at which time the lower end of the esophagus was markedly obstructed by a submucosal or periesophageal constricting ring through which Jackson bougies would curl rather than pass. Because of this marked obstruction, cardiectomy was carried out. Marked improvement followed the surgical procedure and she was discharged on October 28, three weeks after operation.

Her family physician has reported that under stress the patient has dysphagia and regurgitation. Most of the time she was well and she has not had enough difficulty even to desire fluoroscopic examination. The result in this case should be considered only fair.

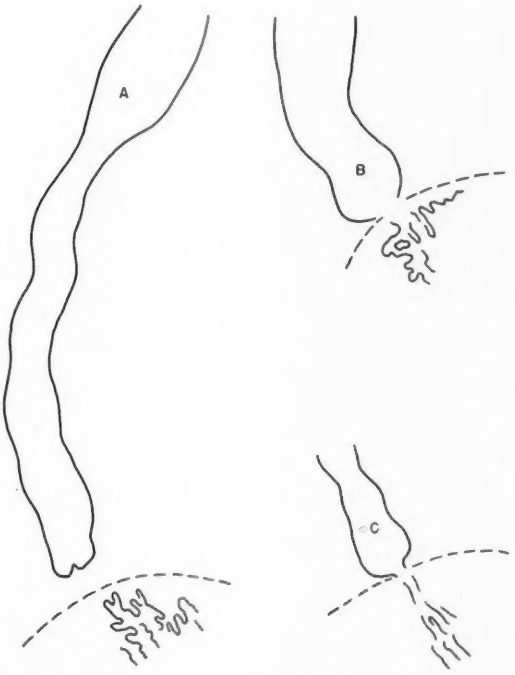

Fig. 5, Case 6.—Outline of barium-filled esophagus showing moderately dilated muscular esophagus, the lumen of which can be occluded by this muscular action.

Pathologic Report: There was marked resistance on attempting to pass the small finger through the lumen of the removed specimen. The specimen was opened longitudinally and measured 5 cm across the esophageal end and 4 cm across the gastric end. It was 7 cm in length, 3.5 cm of which was esophagus and 3.5 cm was stomach (Fig. *7F*). A sharp line separated the esophageal mucosa from the gastric mucosa. The esophageal mucosa was wrinkled and the esophageal wall was 0.7 cm thick.

Microscopic sections showed only slight fibrosis of the muscularis.

The entire thickness of the section was 0.65 cm. The circular muscle coat was twice the normal thickness and the individual fibers of this coat were larger than usual. The longitudinal muscle coat was only slightly thickened, if at all. The muscularis mucosae was thickened approximately 50 per cent. The over-all fibrous tissue content for this section was only slightly increased and was chiefly in the

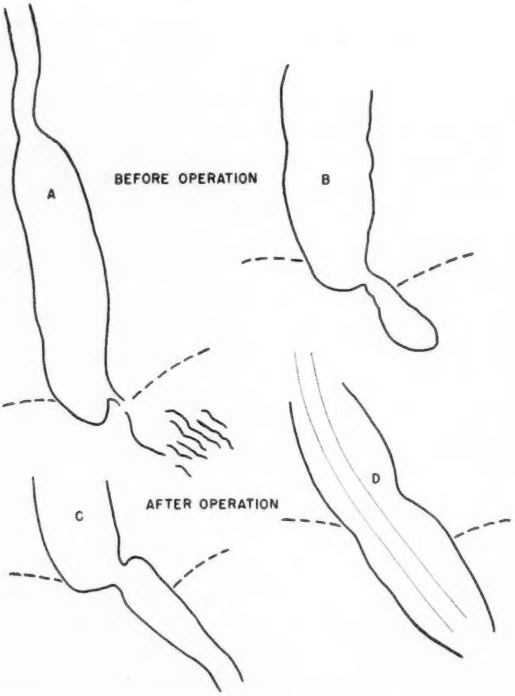

Fig. 6, Case 7.—*A* and *B,* Outline of barium-filled esophagus before cardiectomy; *C,* after cardiectomy; *D,* barium striped dilator in place, distended to 6 pounds pressure.

form of loose fibers between muscle layers. The mucosa and submucosa were infiltrated with lymphocytes to a slight degree but were otherwise negative. Although nerve fibers were abundant, particularly between the circular and longitudinal muscle coats, no ganglion cells were found. Blood vessels and adventitia were negative.

CASE 7.—A woman, aged 27 years, was first seen May 7, 1947, with the complaint of inability to swallow solid foods since the age of 14. She had regurgitated undigested food intermittently and had had retrosternal burning since that time. At the onset of her symptoms she had experienced a severe fright during a thunder storm; following this experience she had been unable to swallow solid foods. She has subsisted on liquids and soft foods exclusively. She often regurgitated foods swallowed six to twelve hours previously. A burning discomfort and a feeling of pressure under the sternum were very distressing, but were relieved occasionally by taking food. Esoph-

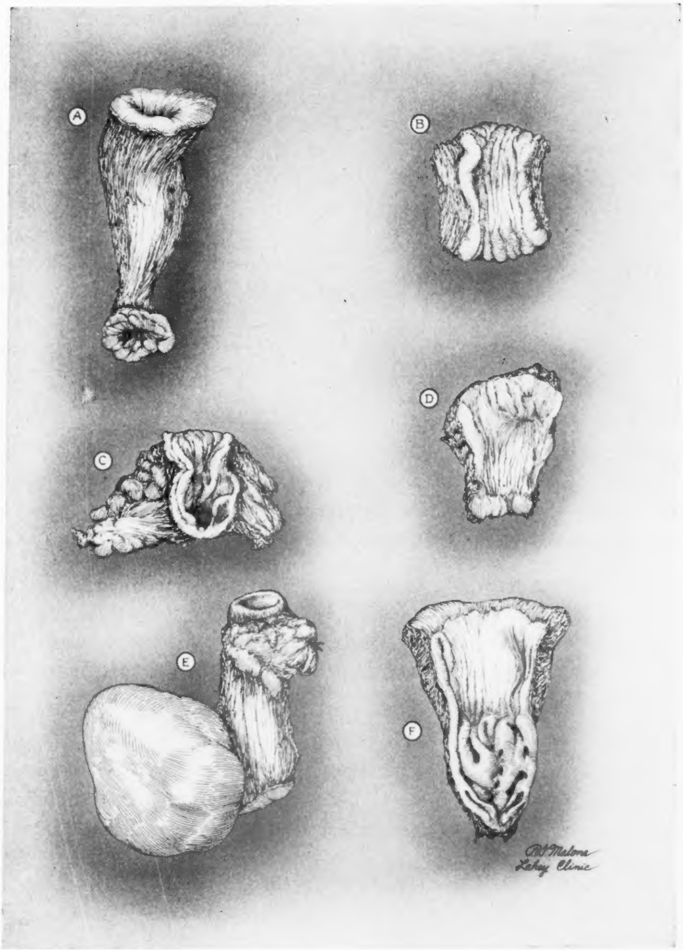

Fig. 7.—*A,* Specimen removed from Case 1; length of specimen 17 cm. See pathologic report, Case 1. *B,* Specimen, 4.5 cm in length, removed from Case 2. See pathologic report, Case 2. *C,* Specimen, 7.3 cm in length, removed from Case 3. See pathologic report, Case 3. *D,* Specimen, 7 cm in length, removed from Case 4. See pathologic report, Case 4. *E,* Specimen, 6 cm in length, with an attached sac 6 cm in diameter, removed from Case 5. See pathologic report, Case 5. *F,* Specimen removed from stomach and esophagus (Case 6). See pathologic report, Case 6.

agoplasty was done elsewhere in December 1941. There was some improvement following this procedure but soon she was only able to take liquids. During the year before she came to the clinic she had been treated with dilations, with some improvement, and had gained 15 pounds.

Fluoroscopy and roentgenograms of the chest and esophagus showed the lungs to be clear. There was moderate dilation of the entire esophagus to a point just above the diaphragm where the esophagus became pouched and constricted but remained smooth in outline (Fig. 6). There was no apparent muscular activity of the lower portion of the esophagus. The barium mixture slowly passed into the stomach. Because of the failure to obtain relief by treatment and because of a history of a previous operation, cardiectomy was recommended. This was carried out on July 10, 1947.

At the time of operation a large amount of scarring was found between the stomach and the lower leaf of the diaphragm and the wall of the esophagus was approximately three times its normal thickness. The lower end of the esophagus was excised and a primary end-to-end anastomosis to the stomach was performed.

The patient was able to eat solid food on July 16, and she was discharged from the hospital July 26, 1947. Her symptoms returned within two months, however, and dilations of the anastomosis were carried out, again with marked improvement. The result in this case must be considered poor.

Pathologic Report: An attempt to pass the little finger through the lumen of the removed specimen tore the stricture or perforated the esophagogastric junction. The specimen when opened longitudinally measured 5 cm by 4 cm, approximately one-half being covered by esophageal mucosa and the remainder by gastric mucosa. There was a 1.5 cm circular perforation. Near the perforation there was a brown, irregular line 3.5 cm long, possibly a scar. The esophageal wall was 0.8 cm thick.

The microscopic examination demonstrated chronic inflammation and fibrosis.

The section was 0.45 cm in thickness, not including the mucosa which was largely necrotic. Both the circular and longitudinal muscle coats were two times the usual thickness. There was a moderate amount of collagenous fibrous tissue between the muscle layers About the blood vessels there was a diffuse infiltration of granulocytes and focal collections of lymphocytes and macrophages throughout the muscle coats. The mucosa was largely necrotic. Nerve

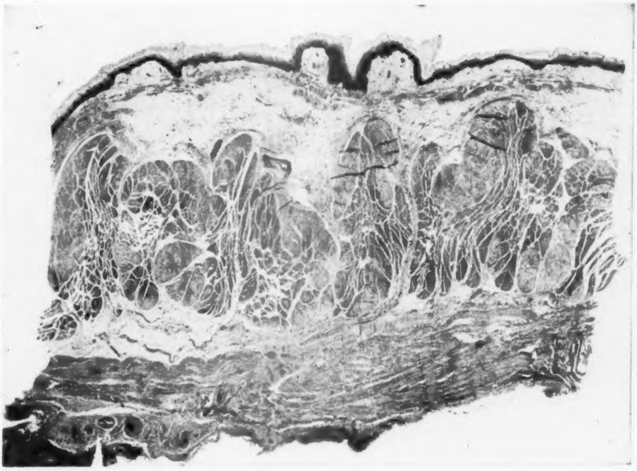

Fig. 8.—Low power view of full section showing marked muscular hypertrophy, especially of the circular (inner) layer. X9

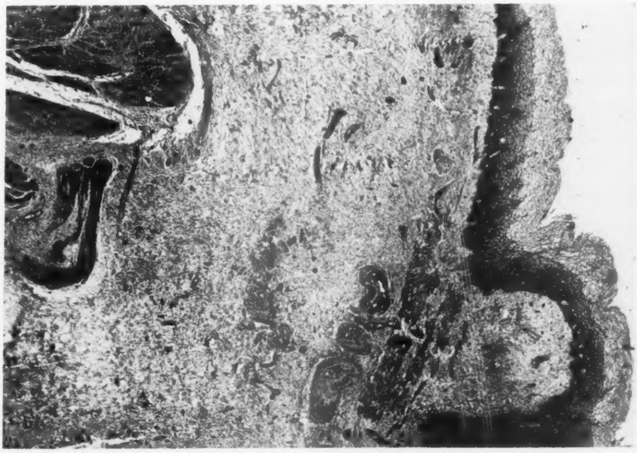

Fig. 9.—Marked widening of submucosa by dence fibrocollagen. X50

fibers were abundant between the muscle layers, but no ganglion cells were found.

SUMMARY OF PATHOLOGIC FINDINGS*

The most striking feature of the pathologic examination was the absence of ganglion cells of Auerbach's plexus in the muscle layers of the wall, although serial sections were not studied. Examinations of random control sections taken of the distal esophagus at autopsy showed numerous ganglion cells in every section examined. The absence of ganglion cells, however, was not associated with an absence, or even a relative decrease, in nerve fibers. On the contrary, it often appeared that there was an increase in nerve fibers between the circular and longitudinal muscle bundles.

A second pathologic feature was the muscular hypertrophy (Fig. 8). The hypertrophy was evidenced not only by the thickness of the respective coats, but also by a thickening of individual muscle fibers. The circular coat of the wall was thickened to the most pronounced degree in all cases, although the muscularis mucosae was often proportionately enlarged. In only two cases did the longitudinal layer take part in the generalized thickening.

Fibrosis was present in all cases and was evidenced by an increase in the amount of connective tissue between individual muscle fibers as well as between muscle bundles and layers (Fig. 8). Often various muscle bundles (Fig. 9) seemed to be largely replaced by collagenous tissue. The fibrosis was particularly pronounced in the subepithelial tissues (Fig. 10) and between the muscularis mucosae. All sections showed a scattering of inflammatory cells. These usually consisted of not much more than the accumulation of lymphocytes and plasma cells which one usually finds in the submucosa of the esophagus. A few cases, however, showed granulocytic infiltration as well and two showed lymphocytes within the epithelium (Fig. 11). In one case there were focal accumulations of inflammatory cells in the muscularis (Fig. 12). The mucosa otherwise was unchanged and showed no unusual thickening of hyperplasia. One case showed epidermidization of gland ducts (Fig. 13). Blood and lymphatic vessels showed no unusual findings in any instance.

From the examination of the various specimens it seems evident that no marked or demonstrable pathologic condition was present in the cases in which the cardia dilated fairly readily and in those in which it did not dilate.

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*The pathologic study was made by Dr. William A. Meissner, Pathologist, New England Deaconess Hospital, and Dr. Robert Prichard.

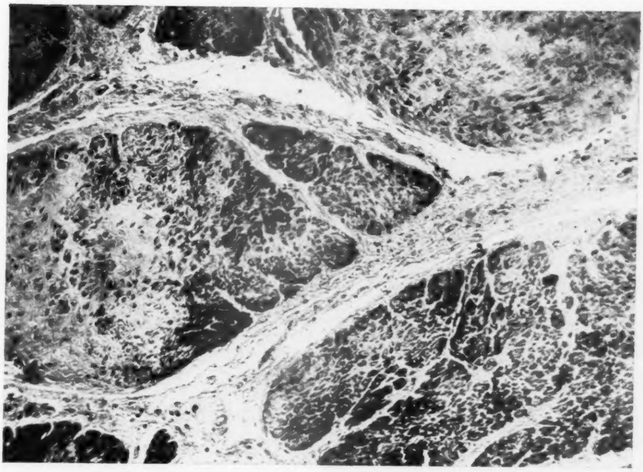

Fig. 10.—Light areas represent fibrous tissue replacing muscle. X130

Grossly, the entire wall of the esophagus was constricted at the esophagogastric junction; the circular muscular coat offered the greatest resistance to the passage of the dilating finger. The circular muscular coat formed the greater bulk of the esophageal wall.

Why does not the circular muscle elongate and the esophageal wall dilate in the region of the cardia as it does above the cardia?

From the fluoroscopic examination of what are thought to be normal esophagi, usually the lumen of the cardiogastric junction remains closed except for the passage of the bolus from the esophagus to the stomach. Frequently, the lower end of the esophagus above the cardia becomes quite distended before the cardia opens. In a normal esophagus I have never observed the cardia to open as widely as the esophageal lumen above the cardia. It seems possible that any infection with fibrosis, inflammation or irritation might make opening the cardia more difficult because of changes in the esophageal wall or increased tone of the musculature in the area, or both. On examination of dilated esophagi irregular dilation often is found, especially about the hilar region. It is my belief that the irregular dilation may occur not only because of an unusual condition of the esophageal wall but because of some extrinsic supporting structure. There is always an extrinsic supporting structure about the esophagus as it extends through the diaphragm before or at the esophagogastric junction.

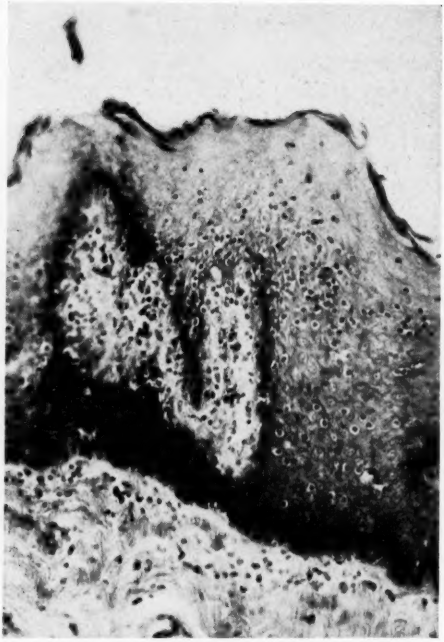

Fig. 11.—Infiltration of mucosa and submucosa by lymphocytes. X350

The muscular wall of any hollow viscus which empties itself by muscular contraction hypertrophies as the result of increased work. This is true of the urinary bladder and of most portions of the gastrointestinal tract. Obstruction to the emptying of the viscus is the most common cause of increased work and is accompanied by hypertrophy of the muscular wall above the obstruction.

As long as the muscular hypertrophy and increased activity of the musculature accomplish adequate emptying of the viscus, the condition may exist for an indefinite time without clinical manifestation of the partial obstruction and the great muscular hypertrophy which has taken place. When the muscular wall of the hollow viscus is no longer able to carry out adequate emptying of the viscus, however, dilation of the hollow viscus takes place and supporting structures either within or without the viscus may alter its shape as dilation takes place. These factors permit an extreme variation in the size and shape of the esophagus.

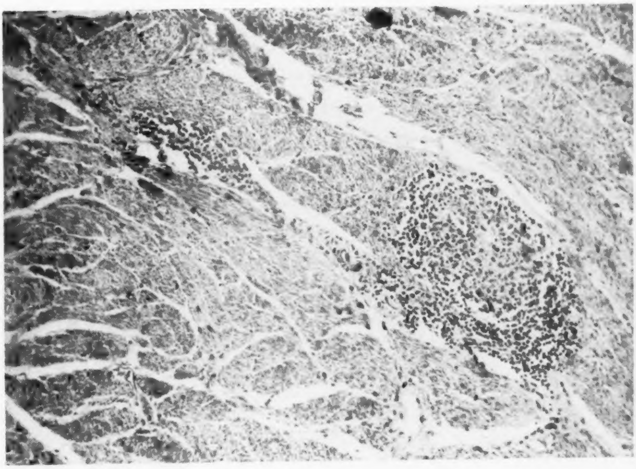

Fig. 12.—Focal collection of lymphocytes and thickened muscularis. X150

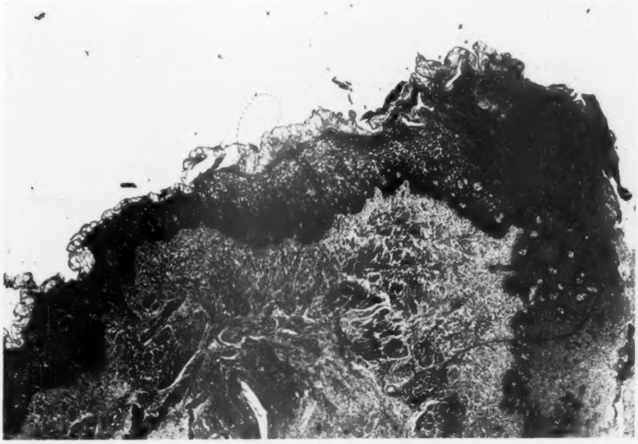

Fig. 13.—Gland in lower center shows epidermidization. X50

TABLE 1.

SUMMARY OF CASES.

CASE	AGE AND SEX	DATE SEEN	AGE AT ONSET	UNUSUAL FINDINGS	DILATION OF CARDIA	SURGICAL PROCEDURE	RESULT
1	47 F	9-47	22	Dyspnea, emaciation	++++	Gastrostomy	Excellent
2	61 F	9-49	21	2 Severe hemorrhages	++++		Excellent
3	42 M	7-47	Indefinite	Dyspnea, weight loss 45 pounds	+		Good
4	68 M	4-47	24-28	Diverticulum, emaciation	++++	Gastrostomy	Excellent
5	69 M	7-48	49	Diverticulum	++++		Fair
6	43 F	9-46	33	Small esophagus	+		Fair
7	27 F	5-47	14	Marked scarring at operation	+	Esophagoplasty elsewhere	Poor

All the specimens showed inflammation and fibrosis. This process alone could produce a stricture or at least resistance to the normal passage of the esophageal content. This resistance in turn produces hypertrophy of the muscular wall. When the resistance is not adequately compensated, the pressure within the viscus produces dilatation of the viscus.

The clinical course of patients with strictures of the lower end of the esophagus varies greatly. A patient may be relatively free from symptoms for a long period without treatment, and for this reason it is difficult accurately to evaluate any therapy. The only treatment on which I rely is dilation of the stricture or its surgical removal.

Failures from dilation, I believe, are due to failure of the operator to apply the proper amount of pressure in the area of the stricture over a sufficiently prolonged time.

Accidents (and I have had 4, or in approximately 0.9 per cent of our cases) have resulted from too great and rapid dilation. Surgical failures have resulted because the stricture was not removed or recurrence of a stricture was not prevented.

I do not advocate cardiectomy as a routine procedure for the relief of stricture of the cardiogastric junction, but point out that there may be suitable cases for its use, as in: (1) an unusually long and tortuous esophagus, (2) when a large diverticulum is also present at the lower end of the esophagus and (3) when a previous operation has failed to relieve the stricture or the stricture is so firm that plastic procedures or esophagogastrostomy may be difficult.

It should also be emphasized that after cardiectomy on a small, thick-walled esophagus, a stricture at the esophagogastric anastomosis may occur from the folds of the tissue at the anastomosis and the contracture of the suture lines. This, however, is unlikely to occur in the markedly dilated esophagus in which a large opening is readily made. The necessity of dilations following cardiectomy emphasizes the fact that this procedure is not entirely satisfactory. Cardiectomy does interfere with the continuity of the alimentary tract and patients who have this procedure have a more difficult postoperative course than those treated by cardioplasty or esophagogastrostomy.

From my experiences with the condition called "cardiospasm," "achalasia," and so forth I believe that such terminology should be abandoned. It should be designated as stricture of the esophagus, the location stated (esophagogastric junction) and any further description added, as dilatation of the esophagus, etiology unknown, or associated with peptic ulcer, gallbladder disease, intracranial disease, pyschic states or any other condition present.

Microscopic study of seven specimens removed surgically has not given information concerning the etiology. The study shows no discernible difference between the stricture that dilates readily and the one which does not.

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DISCUSSION

DR. PAUL C. SAMSON (Oakland, Calif.): I think this is a very interesting and valuable paper. It points up the difficulty we have, not only from the endoscopic, but also from the surgical standpoint. My experience has been limited with this phase insofar as surgery is concerned. We have one patient now on whom we did a resection of the esophagus after an unsuccessful cardioplasty elsewhere, the resection being done primarily because of continued bleeding and ulceration of the lower esophageal segment. We have successfully controlled the bleeding, but I note that Dr. Hoover has had to dilate some of his patients and we are in the process of carrying out dilations on this man.

Very interestingly, the new esophagogastric juncture seems spastic. In front of the fluoroscope the barium comes down the esophagus and sticks, the man's attention is distracted elsewhere and all of a sudden the spastic juncture opens up and barium floods into the stomach. My roentgenologist told me "If I had not known this man had been operated on and had a section of four or five inches of the esophagus taken out, I would say it is still a cardiospasm." I do not know the answer. Certainly this particular operation is not going to solve all of our problems as has been pointed out.

I would like to ask one question. Did Dr. Hoover's surgeon do vagotomies on any of the patients at the time of the esophageal resection?

DR. WALTER B. HOOVER (closing): Referring to Dr. Samson's question concerning vagotomy, I feel that the surgeon made no particular attempt to do a vagotomy. When a vagotomy was done in one case the patient had difficulty emptying the stomach after this procedure. I think that people who have vagotomies for gastric ulcer often have some difficulty in emptying their stomachs and for that reason nearly always a gastroenterostomy is done. Gastroenterostomy was not done in these cases. The patient who had difficulty did clear up in the course of six weeks so her stomach emptied well.

I also wish to point out as Dr. Samson has stressed, the fact that some of these patients reacted after operation similar to a cardiospasm even though the cardia had been removed and the results are not entirely satisfactory. The patient does have less difficulty, but some of them have had to have dilations, and are not entirely relieved of their dysphagia.

LXXVI

HIGH ESOPHAGEAL STENOSIS

CHEVALIER L. JACKSON, M.D.
PHILADELPHIA, PA.

High esophageal stenosis may be due to cicatricial changes following the healing of a corrosive esophagitis due to a burn caused by the swallowing of lye or other caustic alkali or acid. The caustic substance may have been swallowed by accident, as is generally the case with children, or with suicidal intent. On the other hand, high stenosis may be due to the condition generally known as the Plummer-Vinson syndrome. This condition is characterized by stenosis of the cervical esophagus accompanied by glossitis and a hypochromic anemia. High stenosis may also be due to fibrosis of unknown origin. Such cases have been thoroughly studied by Dr. Harris P. Mosher[1] and he believes that stenosis of the cervical esophagus occurs most often as a result of infection in the upper lobes of the lungs or in the pharynx. Carcinoma occurs at this level, of course, as at all other levels in the esophagus, and must always be thought of in the differential diagnosis in the adult.

The difficulties of exposure of the esophageal orifice and the dangers of perforation of the hypopharynx have been well described in the past.[2] The powerful muscular contraction of the cricopharyngeus muscle pulling the cricoid cartilage back against the spine has long been recognized as an obstacle to the introduction of the esophagoscope and has tended to increase greatly the risk of perforation of the pyriform sinuses. The presence of a stenosing lesion at or just below the esophageal introitus, greatly increase the difficulties and the hazards. It has often been stated that the exposure of the esophageal introitus in a patient with pulsion diverticulum of the hypopharynx is extremely difficult without the aid of a previously swallowed string, and in the esophagoscopically guided technic of operation for diverticulum the swallowing of a string previous to the operation has become a routine matter.

As an aid in introducing the esophagoscope into the esophagus with the minimum of risk of perforation, we have long advocated the use of a flexible steel stem filiform bougie as a guide.[2] The scope is passed into the right pyriform sinus, and then the filiform bougie is very gently introduced in such a way that spontaneous

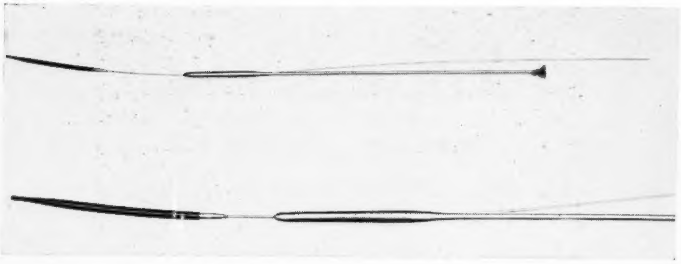

Fig. 1.—Round tracheal dilator passed over steel stem of flexible bougie. These dilators can also be passed over a previously swallowed string, if preferred.

opening of the esophageal orifice is elicited, after which the bougie passes easily down the esophagus and thus serves as a guide for the introduction of the esophagoscope. In the presence of stenosis at this level, obviously, the regular esophagoscope will not advance even if the filiform bougie has gone through, or even if a string has been swallowed as in the case of diverticulum. Sometimes, when confronted with great difficulty in the introduction of an esophagoscope because of high stenosis, the author has passed a filiform bougie as a guide and then endeavored to pass a small esophagoscope over the bougie as a dilator. It has occurred to him, however, that it would be easier and less traumatizing to pass perforated metal bougies over the steel stem of the filiform (Fig. 1). Obviously, a swallowed string can be used in the same way, but this presupposes swallowing the string the previous day, whereas, if a bougie may be passed, the steel stem of the bougie can be used as a guide for subsequent passage of increasing sizes of metallic bougies, without delay. Another instrument that can be used is the small olive tipped dilator passed over a string, or the Plummer dilator with its increasing sizes of olives. We have had success with all of these methods in different cases, but we are at present in the process of designing a telescopically guided dilating esophagoscope to be passed over a string or over the steel stem of a filiform bougie, in the same manner as the metallic bougies are passed, and we believe that it will be even more efficient.

ILLUSTRATIVE CASES

Case 1.—Woman aged 40. This patient has been suffering from a lye stricture since the age of 18 months. As a child she had reached across the table for a can of lye which her mother had put there, presumably out of the child's reach, and she swallowed a

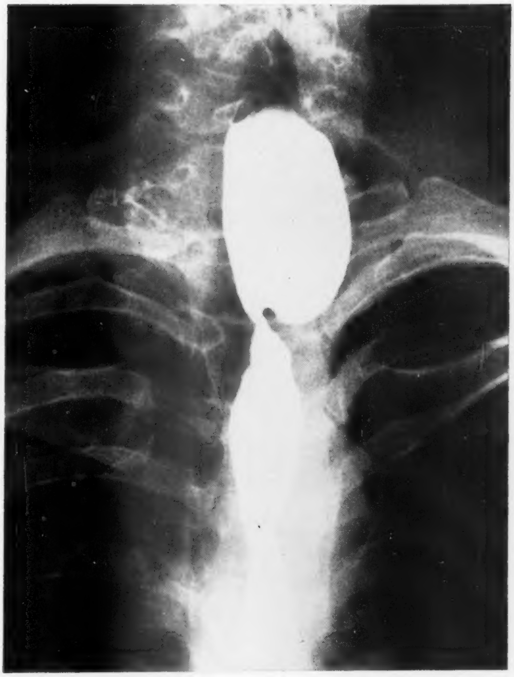

Fig. 2, Case 1.—Stenosis of cervical esophagus due to swallowing of lye in childhood. Because of the great difficulty in accomplishing effective dilatation of this stricture, plastic operation had been thought of, but eventually satisfactory endoscopic dilatation was accomplished.

bit of the lye. Vinegar was given as an antidote and some of the content of the stomach expelled. For several months she was very ill but little medical care was given because the physicians said she would die anyway. Until the age of six she took her nourishment only from a bottle. She took no solid food until the age of 12. During all these years however, she managed to get enough food down to sustain nutrition. When she grew up she trained to be a nurse; this took her five and a half years because she was ill a good deal of the time.

No treatment was given in this case until ten years previous to the time the patient first consulted us. At that time an attempt was made at esophagoscopy and the patient stated that her throat closed for three days. The physician who attempted the esophagoscopy advised gastrotostomy, but she did not have this done. On several

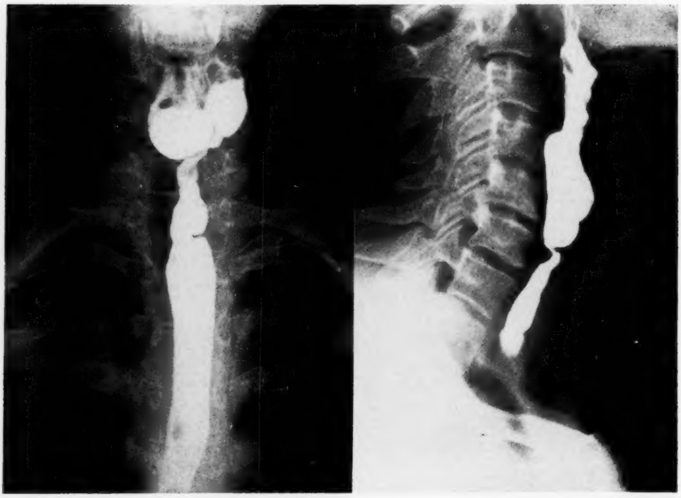

Fig. 3, Case 2.—Stenosis of cervical esophagus due to the ingestion of nitric acid. This stricture proved very difficult to dilate but eventually satisfactory dilatation was accomplished by means of a special technic.

occasions when the esophagus became blocked a rubber tube was passed by a physician and on other occasions by the patient herself, but no effective dilatation was carried out. The patient first consulted us in April of 1945, at the age of 40. During April and May a number of dilatations were accomplished, using a small esophagoscope passed over the steel stemmed flexible bougie, and also some dilatation was done with filiform bougies alone, passed through larger esophagoscopes. It was our conclusion by June 1st however, that very probably a plastic operation should be done with excision of the cicatricial stenosis and anastomosis of the esophagus.

The operation was deferred however, and the patient went home for a time. She returned in October with the report that she had been able to swallow considerably better than before the series of dilatations had been given. Attempts at further dilatation using 3 and 3.5 mm esophagoscopes passed over a swallowed string were successful. On October 23rd, 1945, the stenosis was dilated with a No. 16 Fr. metal dilator passed through a 7 mm full lumen esophagoscope introduced over a string, and subsequently No. 18 to No. 24 perforated bougies were passed over the string. The patient has not been seen since December, 1945, but a very satisfactory report was received from her in May, 1947, at which time she stated that she was swallowing with very little difficulty, though still masticating food carefully. She had gained ten pounds in weight and felt stronger and better than for a number of years (Fig. 2).

Case 2.—Female aged 23. This patient came to us in August, 1937, complaining of an esophageal stricture of ten years duration which followed the drinking of nitric acid in an attempt at suicide. The patient stated that at times she could scarcely swallow anything. She had suffered considerable weight loss, about 40 pounds in a period of several years. Her weight at the time she came to us was 118 pounds. This patient stated that she had been unconscious for two or three days following the ingestion of the acid. Her burns were treated but she was unable to swallow any food on recovery of consciousness. For several weeks she was fed by rectum. Some dilatation was carried out by means of catheters or flexible rubber tubes but nothing else had been done. At our first esophagoscopy it was not possible to pass the 5 mm standard esophagoscope, but a filiform bougie was easily passed. A series of dilatations was carried out using small esophagoscopes and bougies passed through the laryngoscope. On a number of occasions a 3 or 3.5 mm scope was passed over the steel stem of a filiform bougie but it was not possible to pass a larger scope. In May, 1938, after a long series of dilatations, the patient returned to her home in a foreign country. In November 1944, over 5 years later, she returned for another series of treatments and again small esophagoscopes were passed over the stem of the filform bougie. After a series of none too effective dilatations, on November 15, 1949, the stenosis was dilated with a specially perforated No. 18 Fr. metal bougie passed over a string, through the 7 mm full lumen esophagoscope. Subsequently, similar bougies No. 19 and No. 20 Fr. were passed by the same technic. The patient then again returned to her home and at the present time, about six months after the last treatment no report has been received from her as to present condition of her swallowing, but swallowing was considerably improved at the time of her discharge.

SUMMARY

The problem of high stenosis of the esophagus is discussed, especially from the therapeutic viewpoint. Several new technics are suggested for the management of cicatricial strictures at this level. The use of specially perforated round metal dilators passed over a string or the steel stem of a filiform bougie is especially recommended.

3401 N. Broad Street.

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DISCUSSION

Dr. Edwin Broyles (Baltimore, Md.): I would like to ask Dr. Norris one question on this condition of high stenosis of the esophagus. I saw one case about twenty years ago and I have been looking for another one like it. I imagine it is a typical example of the Plummer-Vinson syndrome. This woman had a high stenosis of the esophagus. Barium x-ray showed almost a complete stenosis. She had lost weight and her dysphagia began following a very severe streptococcus throat infection. I thought of course, we were going to find a new growth in this region, but on exposing and lifting the larynx with an esophageal speculum, instead of a growth at this area of the cricoid there was a fine mesh of scar tissue that looked like a miniature fish net. There was no difficulty at all in breaking down these small lesions. This is the only case I have seen. The result was excellent. I want to ask Dr. Norris if he or Dr. Jackson have ever seen a similar case.

Dr. Charles M. Norris (Philadelphia, Pa.): I suppose the case that Dr. Broyles has mentioned might fall into the group which Dr. Mosher has considered a sequel to an infection of the pharynx or upper mediastinum. I do not recall seeing a similar case. It is interesting that the stenosis which developed was easily dilated. In our experience, the only ones in which dramatic relief can be accomplished by one or two dilations are those of the Plummer-Vinson syndrome, where the stenosis is so thin and web-like that there is possibility of restoring a normal caliber of the esophagus by merely passing the esophagoscope through.

LXXVII

HISTOPLASMOSIS—A DEFICIENCY DISEASE

REPORT OF TWO CASES WITH LARYNGEAL INVOLVEMENT

Sam E. Roberts, M.D.

and

Frank S. Forman, M.D.

Kansas City, Mo.

We had not progressed far with our investigation of this interesting disease until we discovered that there were two words in the English language, not commonly used, which were extremely useful.

They proved essential in describing our impressions, some of the reasons for our conclusions, and helped form a basis for a hypothesis.

The two words are serendipity and heuristic. To define serendipity in a satisfactory manner we will have to give the following illustration: When one is intensely studying any one subject, scientific or otherwise, the reading and other investigations often lead into many avenues and paths of fascinating knowledge not anticipated in the original text. What we learn in this manner is called serendipity.

Heuristic means we make a statement we believe factual, but do not have scientific proof. It is made to stimulate investigation and research by others. The first part of this discussion will be strictly conventional and orthodox. The remainder will contain information we obtained by serendipity and presented for its heuristic value.

DEFINITION

Dr. Furolow[1] says: "Histoplasmosis is a subacute to chronic infection caused by the fungus histoplasma capsulatum. It is characterized by a primary, usually benign and widely prevalent form; and a secondary, disseminated, relatively rare and often fatal form. In these characteristics the disease resembles tuberculosis and coccidioidomycosis. The source of histoplasma capsulatum in nature is

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Presented before the American Broncho-Esophagological Association meeting on May 19, 1950, San Francisco. From the Department of Otolaryngology, University of Kansas.

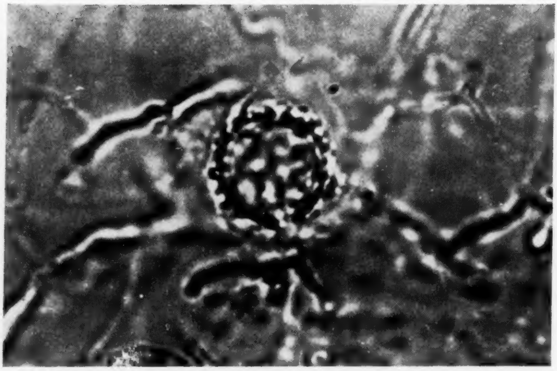

Fig. 1.

not known. The epidemiologic data suggest that it is to be found free in nature but isolation has not been accomplished."

BACKGROUND

First discovered by Darling in the Canal Zone in 1905 who thought the causative agent was a protozoan. Later it proved to be a fungus.

We are reporting on histoplasmosis of the larynx for three reasons: (1) Only 15 cases are reported in the literature involving the larynx. (2) We missed the diagnosis completely in both cases because we did not consider the possibility of histoplasmosis. (3) To give a simplified description of the lesions so our colleagues will be alerted and may make, at least, a provisional diagnosis of this disease clinically and ask the laboratory's help to confirm it. (4) To show an interesting co-relation between the incidence of histoplasmosis and the mineral content of our Kansas soil. (5) To suggest a possible coetiological factor in histoplasmosis and cancer in its geographic distribution. We are going to define the terms commonly used in the description of this disease:

1. Histoplasmosis is the disease.
2. Histoplasma capsulatum is the organism; a fungus.
3. Tuberculate chlamydospores is the name given the spores.
4. Histoplasmin is the testing material; same as tuberculin for tuberculosis.

When we first saw the histoplasma capsulatum (center Fig. 1 x1600) under high power we remarked how much it resembled the insignia of the Rotary Club. This observation has already helped one of our colleagues to identify this fungus. This is a mat of white mycelium which grows rather slowly, requiring one to four weeks for development. On this mycelia mat the characteristic spores develop. These spores are large (15 to 20 microns) have a double refractile wall through which the cell material projects, finger-like.

This fungus, like a number of other pathogenic human fungi (blastomyces, coccidiodies) grows in two phases. The parasitic phase or that which exists in the warm blooded host is a yeast-like organism of small size which is found in the mononuclear cells. The appearance of the yeast-like organism in the cell is characteristic to one acquainted with the pathology of this disease. However, the yeast-like organism in the sputum cannot be differentiated from other common, yeast-like organisms such as monilia. To establish a diagnosis it is necessary to grow the organism in culture. This can be done from sputum, blood, biopsy material, bone marrow or autopsy tissue. In culture at room temperature the organism grows in the second or saprophytic phase. This phase is characterized by growth resembling what is commonly called mold growth.

PATHOLOGY

The organism is found in the:

1. Blood (circulating monocytes).
2. Bone marrow.
3. From granulating lesion removed for biopsy.
4. Sputum-must be cultured.
5. Lymph nodes.
6. Post-mortem material.

Furcolow[1] "Histoplasmosis was formerly thought to be unusually rare and fatal. It is known now to exist as a mild asymptomatic syndrome very prevalent in certain parts of the world. In certain respects the disease so closely resembles tuberculosis as to be frequently confused with it. The most striking similarity of the two diseases lies in the fact that both show pulmonary calcification. Evidence reveals there is little doubt that healing by calcification does take place in histoplasmosis as it does in tuberculosis."

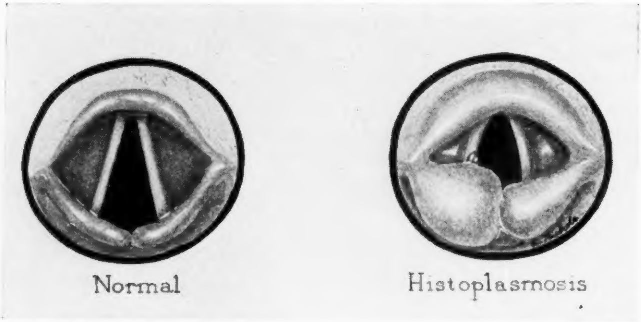

Fig. 2, Case 1.

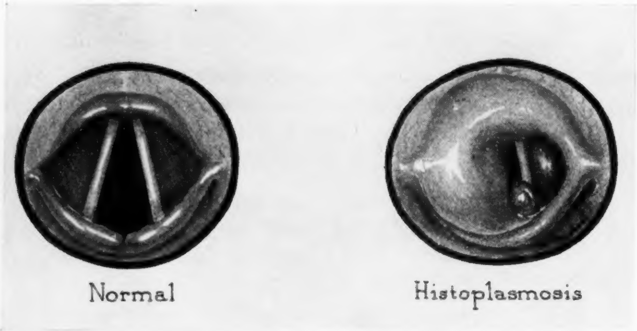

Fig. 3, Case 2.

DIAGNOSIS

1. Positive isolation and identification of histoplasma capsulatum.
2. Positive histoplasmin complement fixation test.
3. Positive histoplasmin skin test.
4. Splenomegaly and hepatomegaly accompanied by leukopenia.
5. Irregular temperature, exhaustion, loss of weight.
6. Usually some pulmonary symptoms; cough, etc.
7. Chest x-ray.

HISTOPLASMOSIS OF THE LARYNX

In both of the patients, we are reporting, the characteristic finding was extreme edema of the entire laryngeal mucosa including the epiglottis. A granulomatous lesion was present in both. Except for the granuloma, the larynx in each patient had the clinical appearance of having been submitted to much recent radiation. One should always be alerted for possible histoplasmosis when extreme edema is encountered in any chronic laryngeal condition with granuloma.

CASE 1.—White male, aged 69. Almost completely aphonic. He had an excellent airway as cords were splinted apart by an enormous interarytenoid edema. He had two direct laryngoscopies elsewhere under local anesthesia. He refused further direct laryngeal examination. Biopsies reported "Chronic Inflammation." He was admitted to the University of Kansas Medical Center December 13, 1949; expired December 27, 1949; cardiac death. Gammell-Breckenridge[2] reported a similar case "lesion produced cord fixation so that airway remained large even on phonation."

CASE 2.—White male, age 51. It is obvious from this drawing that tracheotomy was imminent. We did a direct laryngoscopy and removed the granuloma and made a bronchoscopic inspection of the lower airways. Edema was again the characteristic findings throughout the entire bronchial tree. While in the operating room a tracheotomy was performed. Decannulization in 10 days. Laryngeal and general condition was much improved. The patient is reporting to clinic every 30 days.

Dean[3] describes a similar case: "Epiglottis thick, nodular and pale. False and true cords were involved to such an extent as to be entirely obliterated. Tracheotomy was performed. We are sure Dean means "edema." We did not find a "nodular epiglottis."

Havens[4] reports two patients who had the oral lesions of histoplasmosis. He made the observation that their graulomatous lesions do not bleed when traumatized as do most granulomatous lesions. We have confirmed this observation. That there may be lowered resistance on the part of the body, is born out by Henry Williams[8] in speaking of a group of granulomas: "The pathologist studying sections of the diseased tissues appears to be struck by the absence of the usual signs of resistance to infection. Organisms which are found casually invading the tissues without the customary leukocytic barrier being set up do not appear to be the cause of the lesion."

The above statement by Henry Williams[8] whom we consider a careful observer and an astute clinician gives strength to our hypothesis that histoplasmosis is a deficiency disease.

We are indebted to Dr. Isaac Jones and Dr. Eugene Lewis of Los Angeles for calling our attention to the wise dictum of Jonathan

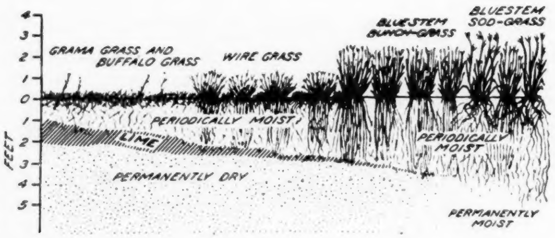
BLUESTEM SOD-GRASS
BLUESTEM BUNCH-GRASS
GRAMA GRASS AND BUFFALO GRASS
WIRE GRASS
PERIODICALLY MOIST
LIME
PERMANENTLY DRY
PERIODICALLY MOIST
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Fig. 4.—(From the Technology Review, Massachusetts Institute of Technology.)

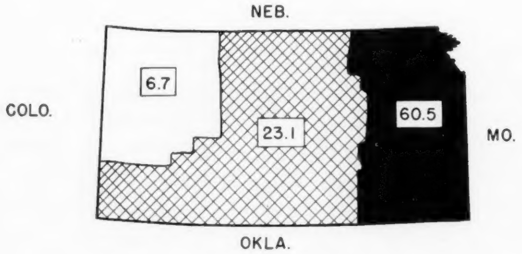

NEB.
COLO.
6.7
23.1
60.5
MO.
OKLA.

Fig. 5.—(Printed through the courtesy of Dr. Michael Furcolow, Surgeon U. S. Public Health Service.)

Wright: "The tubercle bacillus is no less—and no more—the cause of tuberculosis than the skin is the cause of dermatitis. Without the tubercle bacillus one does not have tuberculosis; without the skin one does not have dermatitis.

Also, we have a personal communication from Jones and Lewis as follows: "When Koch discovered the tubercle bacillus, it was believed that it 'produced tuberculosis' but gradually it became apparent that it produced tuberculosis only when the tissues were susceptible. In fact, nearly everybody 'has had tuberculosis,' but disease ceased when nutritional change raised adequate resistance."

We are awaiting with great interest the final publication of the report of Doctor Irene Corey Diller of the Institute for Cancer Research, Philadelphia. It was her opinion as expressed before the American Association for the Advancement of Science that fungi could be a cause of cancer. She reported two types—one altenaria, common in soil and milk, and another syncephalastrum racemosum, common to the tropics.

In the illustrations to follow it will be shown that a higher percentage of the population develops histoplasmosis and certain other degenerative diseases in areas where the lime and other minerals are either too far from the surface of the ground for the plants to reach, or where there is no mineral subsoil at all.

Other illustrations will show the prevalence of degenerative diseases where the soil has been worn out by continuous cropping or erosion, or to many years without minerals.

In Fig. 4 it will be seen that the Western third of Kansas the lime and other elements come within one foot of the surface of the ground. The rainfall is only 17 inches per year. On this soil a gamma grass is grown on which American buffalo grazed many years. The buffalo seldom migrated eastward. There were no fences to stop him. This gamma, or buffalo grass is short, highly proteinaceous, loaded with minerals and trace elements. This was what the buffalo, with true animal instinct, knew was needed for his big bones and a fine body. Where there were many big buffalo there were also, many little buffalo. Propagation was no problem. The wheat grown in this area is famous Kansas hard wheat for which millers pay premiums. Advancing eastward into Central or the middle third of Kansas the lime and minerals are much further from the surface. Rainfall is increased and the grass somewhat taller. In the Eastern third of Kansas the lime has entirely disappeared; rainfall is increased to 37 inches annually and the grass is very tall and appears lush. It is tall, but it is not lush. It is highly carbonaceous and has a minimum of food value for man or beast.

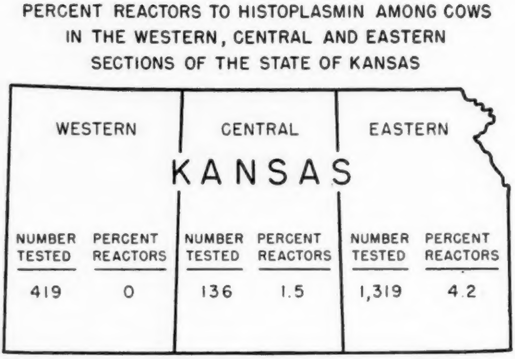
In the Western part of the state, just discussed above, one will see only 6.7% reactors. Central Kansas, where minerals are rapidly receding into the earth the percentage of reactors goes to 23.1%. In Eastern Kansas where lime and minerals have completely disappeared the reactors jump to over 60%.

Fig. 6 is shown through the courtesy of Kansas State Board of Health.

In addition to our statement made in the legend under the map, lower left, we must call attention to the fact that there is only one all white county in the entire Eastern half of Kansas.

Since this article was submitted for publication there has been some very interesting discussion (August, 1950), in Oxford, England, on "The Geography of Cancer," not yet published.

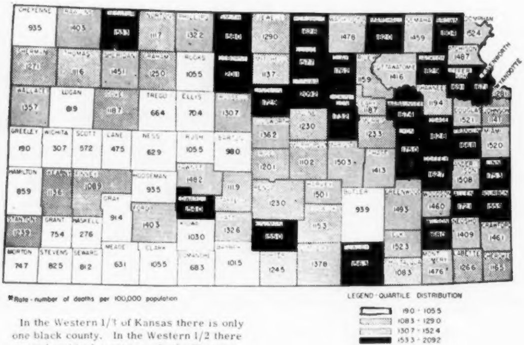
We have carried on a limited, but interesting experimental work with ornamental and fruit trees. The work on the trees was with



PERCENT REACTORS TO HISTOPLASMIN AMONG COWS
IN THE WESTERN, CENTRAL AND EASTERN
SECTIONS OF THE STATE OF KANSAS

WESTERN NUMBER TESTED	WESTERN PERCENT REACTORS	CENTRAL NUMBER TESTED	CENTRAL PERCENT REACTORS	EASTERN NUMBER TESTED	EASTERN PERCENT REACTORS
419	0	136	1.5	1,319	4.2

Fig. 6.—(Courtesy of the Kansas State Board of Health.)



*Rate - number of deaths per 100,000 population

In the Western 1/3 of Kansas there is only one black county. In the Western 1/2 there are only 4 black counties. In the Eastern half there are 21 black counties.

Fig. 7.—(Courtesy of the Kansas State Board of Health.)

the elements found in nails, using ordinary steel nails and galvanized iron nails. Two of each are driven into the base of the tree near the ground. They are driven into opposite sides of the tree so the elements will enter quickly into the tree circulation.

We believe the galvanized iron nail to be the more important, because it is softer, more easily dissolved and oxidized more quickly. Also, it contains more zinc which is important in both plant and animal life. We have found that our ornamental trees have retained their leaves much longer in the fall and the fruit trees develop a better fruit, less apt to fall before thoroughly ripened.

We will report more in detail on this in another year or two.

In addition to the statement made in the legend under the map, we must call attention to the fact that there is only one all white County in the entire Eastern ½ of Kansas.

From the above observations we are going to postulate that histpolasmosis is a deficiency disease, deficient both in minerals and vitamins. We are giving these patients Viterra, made by J. B. Roerig Company of Chicago, which contains 12 trace minerals and elements, as well as multiple vitamins.

VITERRA

Vitamin A (Refined Fish Liver Oil)	5,000 USP U
Vitamin D (Irradiated Ergosterol)	500 USP U
Vitamin B 1 (Thiamine Hydrochloride)	3 mg
Vitamin B2 (Riboflavin)	3 mg
Vitamin B 6 (Pyrodoxine Hydrochloride)	0.5 mg
Niacinamide	15 mg
Vitamin C (Ascorbic Acid)	50 mg
Calcium Pantothenate (Dextro)	5 mg
Mixed Tocopherols Type IV	5 mg

MINERALS

Boron	0.2 mg	Manganese	1 mg
Calcium	213 mg	Magnesium	6 mg
Cobalt	0.1 mg	Molybdenum	0.2 mg
Copper	1 mg	Phosphorus	165 mg
Iodine	0.15 mg	Potassium	5 mg
Iron	10 mg	Zinc	1.2 mg

Nearly a hundred years ago a solution of copper sulphate was painted on the rind of cheese to prevent molds, fungi and attacks by insects.

Those of you who have had experience with fungus attacking plant life know a healthy plant resists fungus disease. Albrecht[5] has shown that brucellosis does not attack healthy animals who have had an adequate nutritional and highly mineralized ration. Allison[6] shows that humans suffering from brucellosis are best treated with minerals; he shows that with proper nutritional background they seldom have the disease.

Francis M. Pottenger[7] gives a most scholarly review of the literature on "The Use of Copper, Cobalt, Manganese and Iodine in the Treatment of Undulant Fever."

Fig. 8 shown through the courtesy of the Kansas State Agricultural College, shows where land is in cultivation 30-35 years and the nitrogen (protein producing element) is reduced from .220% to .125% at Hays, Kansas. It shows it reduced from .120% to almost

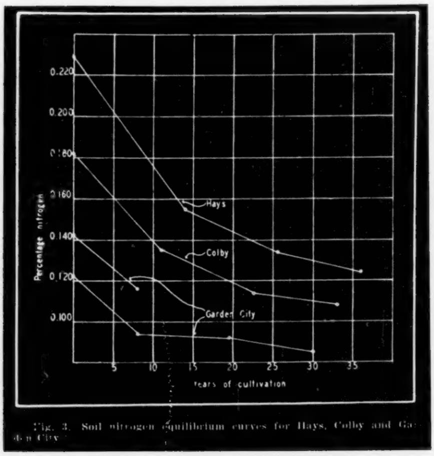


Fig. 3. Soil nitrogen equilibrium curves for Hays, Colby and Garden City.

Fig. 8.

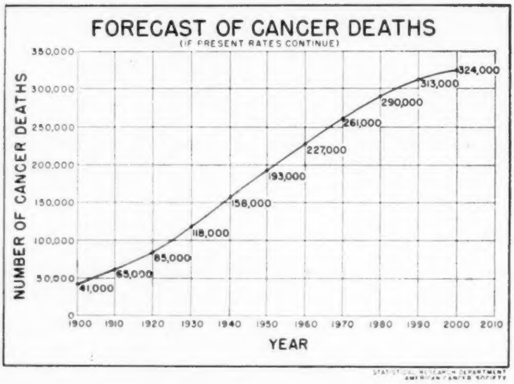


Fig. 9.

zero at Garden City, Kansas. No records are available for other minerals and trace element exhaustion due to years of continuous cultivation, however, it can be concluded logically, that the same destruction is taking place—perhaps, more rapidly. Nitrogen is partly replaced from the air.

The above graph (Fig. 9) is dramatic and self-explanatory.

We are including the two following quotations to show the trend of scientific thinking in recent years:

Doctor Charles F. Nelson:[9] "For the past ten years our research department has been making a study of photomicrographs showing the results of the different deficiencies on most of the tissues of the body—the bone, the retinas, the teeth, all the different glands and all the organs. My associates and I have come to the conclusion that practically every difficulty with the human race has to do with nutrition as it is related to cell chemistry."

Dean J. Roscoe Miller[10] of the Northwestern University Medical School said at the dedication of the National Nutritional Research Center: "Here Spies and his co-workers would be able to pursue a new and more ambitious line of attack, research in the relationship between nutrition and the diseases of middle life and old age—hypertension, cancer, arteriosclerosis and diabetes."

In addition to an adequate nutritional regime we are treating our histoplasmosis patients with Viterra (multiple vitamins, minerals and trace elements). Also, we are treating in the same manner all nerve lesions where we suspect a demyelination of the nerve sheath such as auditory neuritis, ticdouloureux, and other painful head syndromes. All of our patients with malignant disease are receiving Viterra. In addition Magnesium Sulphate 100 mg. t. i. d., and Zinc Sulphate 10 mg, t. i. d., is prescribed. We have no proof of its efficacy. It is our opinion, at this time, that it has been helpful. We are making this statement for its heuristic value only.

CONCLUSIONS

Histoplasmosis occurs in two forms:

1. Mild, benign, asymptomatic and very prevalent in certain parts of the world.
2. Severe, relatively rare and often fatal.

The histoplasma capsulatum (a fungus) must be identified before a positive diagnosis can be made.

It occurs much more frequently in persons who live in areas of the U. S. where soils are most depleted. It occurs more frequently in Central U. S. where seafoods with their abundant mineral supplies are eaten less frequently. In our opinion, while the evidence is not conclusive, it is strongly suggestive that histoplasmosis is a deficiency disease. While there is no specific treatment, it is our opinion that these patients should be given an adequate diet and Viterra, which is a combination of vitamins, minerals and trace elements.

It is also our opinion that cancer and certain kinds of heart and vascular troubles are also deficiency diseases.

It is also our opinion that the fungus histoplasma capsulatum would not attack a healthy person or animal. Since cancer research is a matter of such tragic urgency, we hope the work of Dr. Irene Corey Diller[11] of the Institute of Cancer Research of Philadelphia will be confirmed by others, and a fungus will be found to be the culprit.

We would suggest that every physician read, "The Healthy Hunzas" by J. I. Rodale, and Alton Oschner's article on "The Influence of Serendipity on Medicine," Oschner Clinic Bulletin, June 30, 1946.

1110 PROFESSIONAL BUILDING.

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DISCUSSION

DR. LOUIS CLERF (Philadelphia, Pa.): I became interested in this condition, as one obviously would after stumbling across a case working on it for some time, and finally finding out what the answer really is. Two cases that we had the opportunity to observe have been reported in the literature by Gammell and Breckenridge. One came because of hoarseness, and the second patient had been under treatment in the dermatology outpatient department. They made a diagnosis of sarcoidosis, and referred her for study of hoarseness. We concluded the laryngeal lesion was tuberculosis but the final diagnosis was histoplasmosis. The lesions in the larynx were sufficiently typical to warrant one to make a provisional diagnosis of tuberculosis. There was no evidence of pulmonary tuberculosis and the biopsy report was granuloma.

In spite of all forms of therapy both patients died, and that provided the opportunity to study both at postmortem.

Although it is a systemic disease it was strange that in both of these cases there was originally laryngeal involvement. While there was regression of the laryngeal lesion in the second case with no clinical evidence of laryngeal disease at the time of death, in the first case there was extension of the laryngeal lesion to the lower trachea. While both exhibited the characteristic findings in the adrenal glands one had extensive lesions in the lung and extensive ulceration in the colon.

I think this is an interesting clinical entity and we ought to be keenly alive to it as a diagnostic possibility, and also as another systemic disease that has manifestations in our particular field.

Dr. Paul H. Holinger (Chicago, Ill.): As Dr. Roberts mentioned, histoplasmosis is a systemic disease of fungus origin. Our case which we reported in 1941 is in the medical literature, not in the laryngological literature, and therefore, not in the reports of the otolaryngologists. We found a tumor in the posterior commissure of the larynx of our patient, and in deference to Dr. Roberts, there was no edema of the larynx at any time. The patient had been hoarse and was referred because of the presence of a tumor in the larynx. A histologic diagnosis was made on the biopsy material and confirmed by tissue cultures of the material from the larynx itself, in this case the first positive diagnosis of histoplasmosis made in the living patient. From the cultures of material taken at that time, the original skin test material was prepared. The studies which subsequently have been made so extensively in Kansas followed and the widespread evidence of histoplasmosis was found. Dr. Rigby working with Dr. Biggs at St. Luke's Hospital in Chicago examined a number of children with calcified areas in the lung who had negative tuberculin tests; these were found positive for histoplasmosis.

The disease has two characteristics, one in which the pulmonary lesion apparently heals; the other in which the systemic involvement is more extensive and is generally characterized by an involvement of the adrenal gland. As Dr. LeJeune, Dr. Clerf and Dr. Roberts said, the similarity between this disease and tuberculosis is constant and particularly so with the appearance of Addison's disease in these patients. I think that therapy at the present time should be directed toward the correction of the Addison's disease and adrenal insufficiency with some of the newer compounds in addition to the anti-fungus materials, which unfortunately seem to have little effect on histoplasmosis.

Dr. Francis LeJeune (New Orleans, La.): Dr. Roberts briefly mentioned the word "serendipity." I would like to refer those interested to an article written several years ago by Dr. Alton Ochsner on serendipity in medicine in which he defined this word as the accidental discovery of something important while searching for something else. Thus, one well known example is penicillin.

I was particularly interested in Dr. Roberts' report of two cases of histoplasmosis of the larynx. In July 1947 I saw a man, 44 years old, with a lesion on one vocal cord which proved to be histoplasmosis. He had been hoarse for six months and clinical examination revealed a lesion involving the entire right vocal cord. The cord was slightly reddish in color, thickened in its entirety but more so in its middle third, and finely granular in appearance. Because I was convinced of the existence of a malignancy, I advised a rush biopsy to be followed by laryngofissure. The biopsy was done and the pathologist proved my suspicion to be absolutely wrong by informing me that the lesion was histoplasmosis. Naturally, the operative procedure terminated immediately and in the next few days I acquired considerable information on the subject of histoplasmosis. The appearance and location of the lesion involving the vocal cord in my patient differed considerably from the two described by Dr. Roberts. I am convinced that until more clinical data are obtained on such cases, a diagnosis of primary histoplasmosis of the larynx will be extremely difficult to make. An accurate diagnosis can be made only by biopsy.

My patient was given 4 mg of promine intravenously three times a day and a laryngeal spray of 5 mg of promine every three hours. The infection within the larynx promptly subsided, but the patients now has evidences of histoplasmosis of the adrenal. I have examined him on numerous occasions and I am happy to report that the laryngeal lesion has been completely absent since October 1947. I last saw the patient March 5, 1950. He has a copper colored skin, has evidence of Addison's disease, tires easily, but maintains his weight, and has a perfectly normal voice with normal appearing cords.

I would like to show two slides from a frame of 16 mm motion picture of my patient. Although this picture is small, it does show the thickening and granular appearance of the lesion. There was no apparent interference of motion of the vocal cords.

The other slide shows the area from which a biopsy specimen was taken. A generous bite was obtained with the laryngeal basket forceps, as laryngofissure was contemplated. It is amazing to see how repair has taken place so that today the cord appears normal. Dr. Roberts has shown that histoplasmosis in general is more prevalent in certain areas so that we should all be on the alert for such cases.

Dr. Sam E. Roberts (closing): I want to thank Dr. Clerf, Dr. LeJeune and Dr. Holinger for their discussion. We have every reason to believe the second patient will recover. We were able to decannulate him in ten days. His edema subsided and there was no recurrence of his granuloma at the end of three or four months.

The geographic distribution of this disease and cancer is quite interesting. While the information is in no way conclusive, it is certainly suggestive that they are both deficiency diseases.

LXXVIII

THE DIAGNOSIS AND TREATMENT OF BENIGN STENOSIS OF THE ESOPHAGUS, WITH SPECIAL REFERENCE TO TREATMENT BY DILATATION WITH THE INDWELLING CANNULATED BOUGIE

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AND

(by invitation)

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The diagnosis of benign stenosis of the esophagus is established by x-ray study, visualization of the lesion by esophagoscopy and biopsy in certain cases to exclude malignancy. The treatment depends upon the cause of the lesion and the degree of stenosis. In this paper we are presenting the clinical results so far obtained with the Tucker indwelling bougie which has been described previously (Fig. 1).

Fourteen cases were observed and treated in the Department of Bronchology, Esophagology and Laryngeal Surgery of the Graduate Hospital, with the assistance of Drs. Lautz, Tracy and Fearon. Modifications and adaptations of the indwelling bougie have been presented at a previous meeting of this Association and published in its Transactions.

Caustic Burns. The most frequent type of stenosis is that due to caustic burns. In acute burns with edema of the mucous membrane, the administration of antibiotics in the early stages to con-

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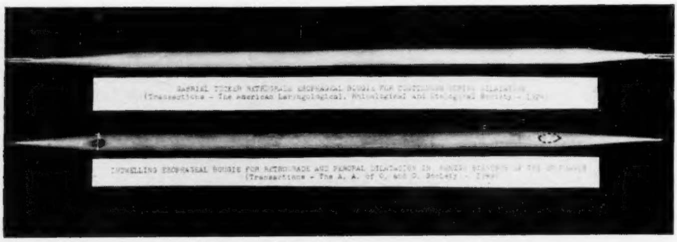

Fig. 1.—Previously presented esophageal bougies. (Modifications of the indwelling bougie published elsewhere in the 1950 Transactions of the American Broncho-Esophagological Association.)

trol infection and ulceration is advisable. Trauma to the burned esophagus should be avoided. Administration of the chemical antidote and removal of the excess of the caustic by aspiration and lavage is the first emergency treatment.

The extent of the burn is best determined by x-ray study with an opaque mixture. Lipiodol is preferable in the examination of children and is introduced through a small catheter which is passed into the esophagus below the level of the cricopharyngeus. The oil is introduced with the patient in the recumbent position. With a small amount of lipiodol the contour of the esophagus can be determined readily indicating the location and severity of the burn. Tube feeding may be used, employing a small catheter adapted to the size of the esophagus, the tube being removed after each feeding. If ulceration is not active indwelling feeding tubes remaining in the esophagus for a period of a few hours, may be used and appropriately sized mercury bougies may be passed to maintain the esophageal lumen and aid in reducing the swelling and inflammatory reaction. When the acute edema has subsided, repeated x-ray examination and esophagoscopy determine, definitely, the location of the stenosis (Fig. 2*a* and *b*).

The Indwelling Bougie. The indwelling bougie may be placed in the esophagus at the time cicatrization is developing. The length of the bougie is such that it will pass well into the stomach of a child; the short bougie may be used in infants. The upper end should be suspended in the esophagus so that the slender tip and suspension string only are above the cricopharyngeus. Severe stenosis seldom occurs above the level of the suprasternal notch, and the opening in the bougie permits fluids from the mouth to pass into the stomach through the strictured area when the bougie is properly placed (Fig.

2*c*). The bougie should remain in the stenosed area six to twelve hours, during which time fluids are given and pass through the cannulated bougie. The size of the bougie is increased as the stenosis recedes. The lumen through the stenosis is determined by the use of an opaque oil and roentgenographs. If the oil passes around the outside of the bougie, the next larger size is used at the next treatment (Fig. 5*c*). The sizes are stepped up until the esophagus is healed and the desired lumen obtained. The complication that has been most troublesome with all methods of indwelling has been serious injury to the larynx, due to the pressure of the indwelling tube on the larynx at the level of the cricopharyngeus. When the cannulated bougie is properly placed, there should be little danger of this complication because only the tip of the bougie is in the hypopharynx. Among the various attempts that have been made to prevent laryngeal complications by avoiding pressure on the larynx has been the introduction of a feeding tube through an external esophagotomy as practiced by Belinoff[2] and Leegard.[6] Lagergard[5] devised a special intubation tube with grooves on the outside of it so that saliva could pass through the stenosis outside the tube. The Tucker cannulated bougie allows the saliva to pass through the central canal of the indwelling tube, and as the stenosis is dilated, saliva also passes around the tube.

Gastrostomy. In severe burns, a properly performed gastrostomy carries practically no risk, puts the esophagus at rest and provides the best means of giving the patient adequate nourishment. Following gastrostomy, the early placement of a string by the string-swallowing method of Mixter[7] should be done. With a continuous string in position, the indwelling bougie may be placed in situs either retrograde or perorally. The indwelling bougie is left in position six to twelve hours in gastrostomized patients two or three times a week. The following case illustrates the management of the acute caustic burn.

ACUTE CAUSTIC BURN OF THE ESOPHAGUS

CASE 1.—A male, 23 months old, swallowed a lye solution. Emergency treatment was instituted and the extent of the burn was determined by roentgen examination. Tube feeding and dilatation of the esophagus in the acute stage were carried out. Cicatricial stenosis developed at the level of the suprasternal notch (Fig. 2*b*). A gastrostomy was done, a string placed in the esophagus and satisfactory dilatation was obtained by the use of the cannulated indwelling bougie. At the end of six months the patient was taking food normally and feeding was carried out entirely by mouth. The

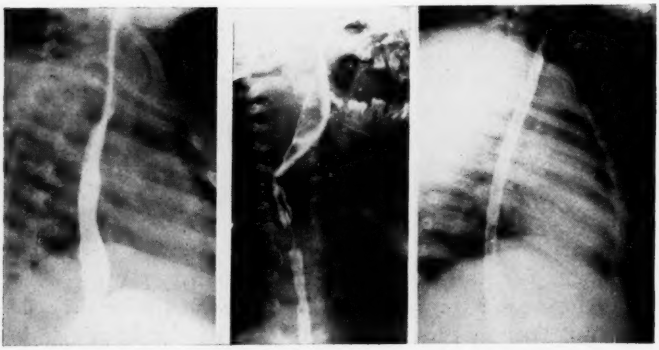

Fig. 2, Case 1.—*a.* Radiogram showing opaque mixture introduced through a nasal catheter outlining the esophageal lumen in an acute burn. *b.* Catheter through nasopharynx outlining cicatricial structure at the suprasternal notch. *c.* Indwelling cannulated bougie properly placed through the cicatricial stricture level. The tip of the bougie only is in the pharynx.

child has regained a normal weight for his age without complications (Fig. 2*a*, *b* and *c*).

CHRONIC CICATRICIAL STENOSIS

A group of patients who had chronic cicatricial stenosis that had failed to respond to various methods of dilatation has been treated. In these cases, the poor result was due to the failure of the patient to cooperate by neglecting or refusing to appear at proper intervals for treatment. In other instances the scar could not be dilated by the method employed. In this group the use of the indwelling cannulated bougie has proved most effective. A closely fitted cannulated bougie placed in a chronic stricture for a period of 24 to 48 hours has not failed to dilate the stenosed area from two to four bougie sizes, thus permitting the insertion of a larger bougie. During this period fluids were passing from the mouth into the stomach through the bougie, and the patients suffered no ill effects from the sojourn of the bougie. The cases observed were: chronic stenosis following caustic burns, stenosis resulting from a congenitally short esophagus, and a peptic ulcer of the esophagus where the scar tissue had resulted from chronic ulceration. In one case following thyroidectomy and postoperative irradiation for carcinoma of the thyroid, chronic stenosis (and finally complete atresia) developed at the level of the cricopharyngeus. This patient had been under observation and treatment for ten years. The use of the cannulated bougie, after perforation by cutting through the scar

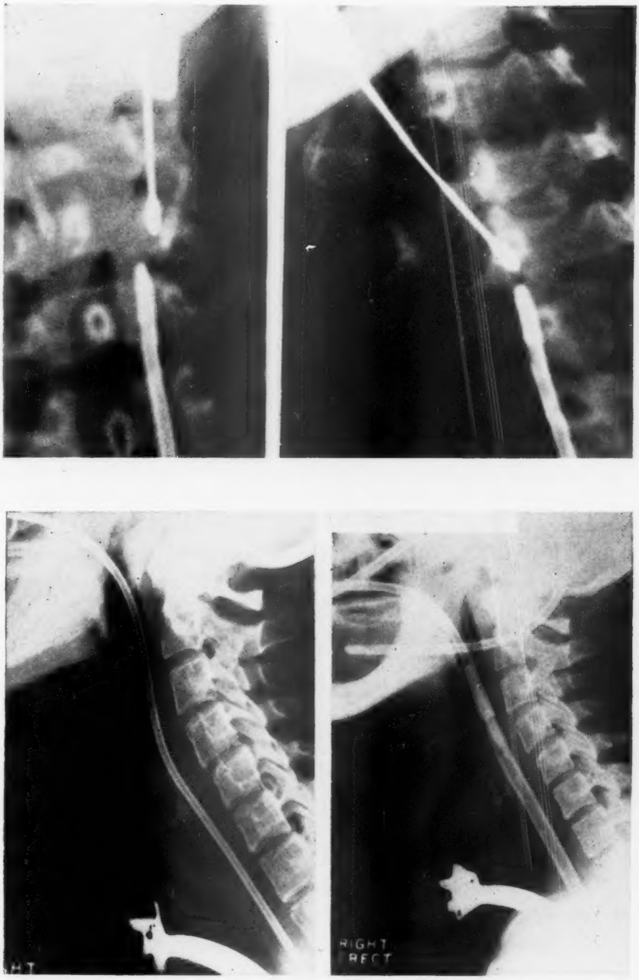

Fig. 3a, Case 2.—Showing metallic bougie introduced from above through a laryngoscope to the upper level of the atresia. Opaque catheter introduced through the gastrostomy opening into the cervical esophagus, showing the lower level of the cicatricial atresia.

Fig. 3b.—Lateral film of figure 3a showing the extent of the cicatricial atresia.

Fig. 3*c*.—Lateral film showing No. 18 catheter in place after incision through the fibrous atresia.

Fig 3d.—No. 18 Fr. catheter has been replaced by the Tucker indwelling bougie. The fenestration in the bougie is seen in the pyriform sinus at the level of the arytenoid.

at the level of the atresia, has resulted in the restoration of the normal lumen at the level of the cricopharyngeus.

POSTIRRADIATION STENOSIS OF THE ESOPHAGUS

COMPLETE ATRESIA AT THE LEVEL OF THE CRICOPHARYNGEUS

CASE 2.—L. C., female, aged 40. In this case complete atresia of the esophagus at the level of cricoid cartilage was treated by external esophagotomy and the placing of a cannulated indwelling bougie from the pharynx through the scar tissue at the level of the cricopharyngeus. The patient had a history of difficulty following a thyroidectomy and postoperative irradiation for carcinoma of the thyroid at the age of 30. A bilateral posticus paralysis developed which required tracheotomy. Ulceration of the esophagus with stenosis of the esophagus necessitated gastrostomy. The stenosis of the esophagus was relieved temporarily by the dilatation and showed considerable improvement, and the gastrostomy was allowed to close. The improvement, however, was not permanent. Stenosis again developed and a second gastostomy was done. Attempts at dilatation were discontinued and the patient was referred to my service at the Graduate Hospital. Our examination revealed a complete atresia of the esophagus at the level of the cricoid. The patient was wearing a tracheotomy tube, and with this seemed to have an adequate airway and a good voice while wearing a valvular tube.

Procedure: By combined examination from the pharynx above and retrograde examination through a gastrostomy fistula we were able to demonstrate the stenotic area at the level of the cricoid. The scar formation was very firm and completely obliterated the lumen at this level for about 6 mm (Fig. 3 *a* and *b*). The extent of the atresia is well demonstrated on both anteroposterior and lateral films. Several attempts had been made at relief of the stenosis by external esophagotomy before her admission. The neck showed extensive scarring over the sternomastoid muscle below the level of the cricoid from these unsuccessful procedures. Dr. Herbert Reid Hawthorne performed an external esophagotomy and was able to approach the stenosis from below. With transillumination through a direct laryngoscope in the hypopharynx, the fibrous atresia was cut through behind the cricoid into the esophageal lumen. A Mathieu forcep was passed through the incision from above and the open end of a No. 18 Fr. Levin tube was passed through the esophagotomy and pulled upward from the esophagus into the pharynx. The perforated end of the tube was passed downward through the cervical and thoracic esophagus into the stomach and a continuous string was washed through the feeding tube from its upper end which had been brought

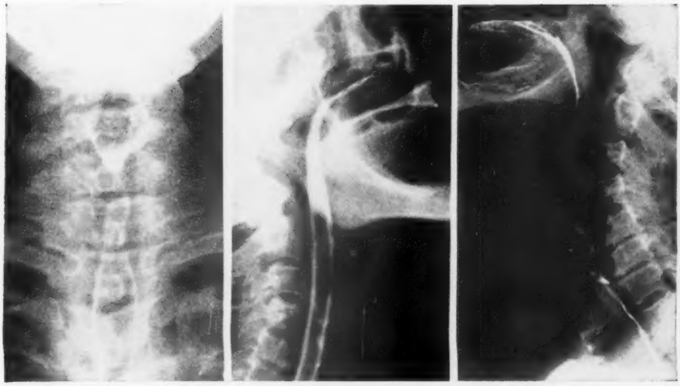

Fig. 4, Case 3).—*a.* Anteroposterior film showing area of stenosis in the cervical esophagus. *b.* Lateral film showing Tucker indwelling bougie (No. 32 Fr.) through the strictured area. *c.* Lateral film of the neck showing opaque mixture passing normally through the strictured area of the esophagus. Patient had gained 25 pounds in a period of two months.

through the anterior nares. This string was recovered from the gastrostomy fistula and was later used to introduce the Tucker indwelling bougie from above. Dr. Hawthorne closed the external esophagotomy and the incision in the neck and the wound healed without infection. The No. 18 Fr. Levin tube was allowed to remain in position for a period of ten days. The saliva, for the first five days, was removed by means of a Stedman pump; on the fifth day the feeding tube was fenestrated above the level of the cricoid so that the saliva could pass from the pharynx through the Levin tube into the stomach. At the end of ten days the Levin tube was brought from the nasopharynx through the mouth and with the aid of the previously introduced string, the tip of a No. 18 cannulated bougie was pulled into the open end of the Levin tube. Then the Levin tube was removed downward through the stomach and the cannulated bougie drawn into such a position that the upper fenestration of the cannulated bougie was in the pharynx and the fenestration of the lower end in the esophagus in the former place of the Levin tube. The saliva could now pass from the pharynx through the cannulated bougie into the stomach. The patient was quite comfortable with the bougie in position; the bougie sizes were stepped up as the opening through the stenosed area enlarged. The bougies were kept in position continuously until a No. 20 Fr. cannulated indwelling bougie had been placed. After 24 hours the increasing sizes of cannulated bougies were introduced from above every third day with the continuous string and allowed to remain in position for 12 hour periods until a No. 32 bougie was reached. Following this, the patient was able to swallow fluids with the continuous string in position and dilatation was continued by pulling the cannulated bougie downward intermittently, until the lumen had reached a No. 38. The dilatations were then continued by the patient swallowing mercury bougies in increasing sizes. At the end of a six months period the patient was able to swallow a No. 40 mercury bougie and was taking all types of food by mouth normally and the gastrostomy opening was ready for surgical closure (Fig. 3).

CHRONIC STENOSIS OF THE ESOPHAGUS

PLUMMER-VINSON SYNDROME

Case 3.—In another case of chronic stenosis of the esophagus, a female 70 years of age (with the typical findings of a Plummer-Vinson syndrome) developed severe dysphagia and lost 20 pounds in weight. Esophagoscopic dilatation and mercury bouginage gave only temporary relief. An indwelling bougie was introduced perorally (No. 18) for a period of 24 hours after direct laryngoscopic dilatation. Four days later a No. 24 Fr. bougie was left in position for eight hours. After another four days a No. 32 cannulated bougie remained for eight hours in the cervical esophagus. Following this, a 7 mm esophagoscope passed though the formerly stenosed area with normal resistance. During the past six months the patient has had no further dysphagia and takes both liquids and solids without difficulty and has gained 25 pounds in weight (Fig. 4).

CHRONIC CICATRICIAL STRICTURE. OLD LYE BURN

Case 4.—D. C., age 15, male. The patient had swallowed lye when five years of age. A severe burn of the esophagus with deep ulceration resulted. Gastrostomy was done. A string was placed and continuous string retrograde dilatation carried out during a ten year period with poor results. The stricture in the esophagus would admit only a No. 18 or No. 20 Fr. bougie. The patient was very uncooperative, resisting treatment, and would not come in for observation.

Mercury bouginage was attempted but was not successful. Surgical resection of the strictured area in the esophagus was considered but the consent of the parents could not be obtained for this procedure. Indwelling bougies were used, beginning 12-13-49, starting with a No. 18 Fr. and allowing the bougie to remain in for a 24 hour period. The sizes were gradually increased until the esophageal lumen was dilated to a No. 34 Fr. Mercury bouginage was again

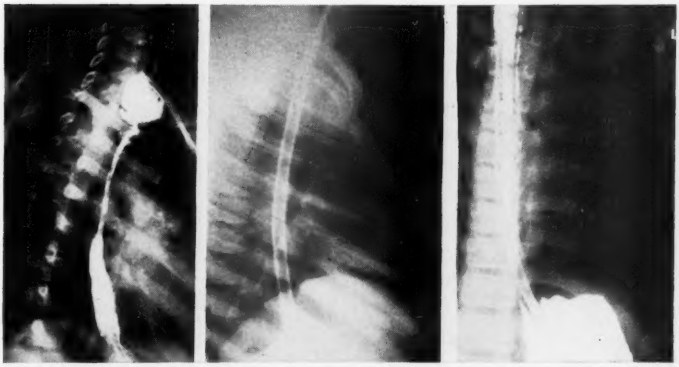

Fig. 5, Case 4.—Old lye burn of the esophagus. *a*. Lateral film showing area of stenosis and dilated esophagus at and above the level of the supra-sternal notch. *b*. Lateral film showing No. 34 Fr. Mercury bougie through the stenosed area of the esophagus. *c*. Film showing latex indwelling catheter No. 28 with the opaque mixture passing through and around the indwelling catheter.

attempted but was unsuccessful in maintaining the esophageal lumen. The stenosis recurred. An indwelling latex catheter, size 24, was placed in the esophagus for one week and replaced with a size 28. The catheter introduced from above was brought out through the gastrostomy opening and was fenestrated in the portion passing through the stomach so that the patient could be fed through the gastrostomy and also take liquids by mouth through the same tube. (The catheter was described in the instrument presentation at the 1950 meeting of the Broncho-Esophagological Association). The catheter was held in the esophagus by suspension from a string attached to its upper end, brought out through the nasopharynx. The funnel shaped portion of the distal end of the catheter was placed above the strictured area and could be withdrawn through the mouth without pulling it through the stenosis. This instrument was in position at the time that this report was made. Treatment is not complete and further progress of the case will be reported later (Fig. 5).

CHRONIC STENOSIS DUE TO SHORT ESOPHAGUS (CONGENITAL)

CASE 5.—F. M., female, white, 25 years of age. This patient first came under observation at ten years of age at another hospital. Esophagoscopic examination revealed a short esophagus with ulceration at the esophagogastric junction at the lower end of the middle third of the esophagus. This diagnosis was based on the x-ray findings and the typical endoscopic appearance of a cicatricial stenosis with an overlying pseudomembranous whitish exudate which left a raw, bleeding surface on removal of the pseudomembrane. The child was treated for a period of several months by peroral esophagoscopic dilatation and local application to the ulcerated area. She was relieved and failed to return for further observation and treatment. Twelve years later at the age of 22 she was again observed at the clinic of the Department of Bronchology, Esophagology and Laryngeal Surgery at the Graduate Hospital. During this interval she had had almost continual difficulty in swallowing and occasional complete obstruction of the esophagus. In spite of this she was well nourished and well developed. Esophagoscopic examination showed a polypoid mass obstructing the lumen through the stenosed area at the level of the junction of the lower and middle thirds of the esophagus. Tissue removed on histologic examination showed inflamed gastric mucosa confirming the diagnosis of congenital short esophagus with cicatricial stenosis.

The patient was treated by peroral esophagoscopic dilatation and local applications through the esophagoscope to the ulcerated area of the esophagus. She improved under this treatment and came in occasionally for dilatation by mercury bougies. She married and bore a child during this interval of observation. The stenosis persisted. A No. 18 Levin tube was passed after dilatation and was allowed to remain for 24 hours in the esophagus. An opaque mixture was given with the dilating tube in position and we were able to demonstrate the passage of the barium around the indwelling tube into the stomach. The lumen could not be increased by this method beyond a size 20 mercury bougie.

Treatment with the indwelling cannulated bougie was begun March 2, 1950. A No. 18 cannulated bougie was introduced for a period of four hours, followed by a No. 24 and No. 26 for twelve hours each at intervals of two days. This was continued until a No. 30 passed easily through the strictured area. The patient is still under observation, has no difficulty swallowing, has gained 10 pounds in weight and at the present time readily swallows a No. 36 bougie (as an out-patient). These sizes will be stepped up until a No. 40 bougie passes with only normal resistance.

This patient did not have a gastrostomy at any time. The indwelling tubes were introduced perorally (Fig. 6).

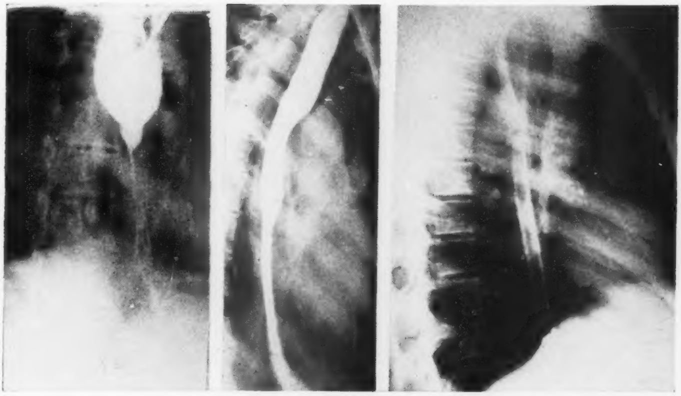

Fig. 6, Case 5.—Congenital short esophagus. *a.* Film showing cicatricial stricture at the junction of the middle and lower thirds of the esophagus. Stomach rugae above the level of the diaphragm. *b.* No. 18 Fr. catheter passed into the stomach with opaque mixture passing through the stricture around the catheter. *c.* Indwelling cannulated bougie, No. 26 Fr., through the strictured area. Patient can now swallow a No. 40 Mercury bougie. Gastrostomy was not required in this patient.

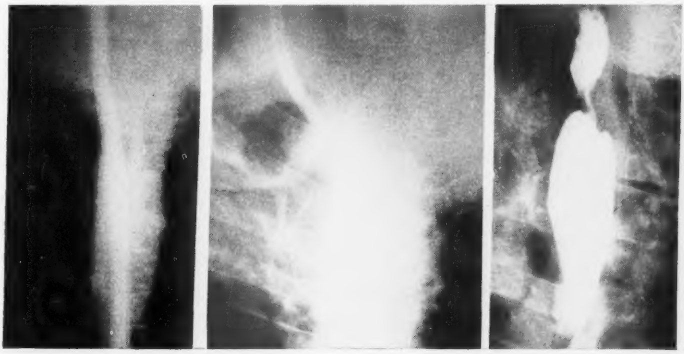

Fig. 7, Case 6.—Beign stenosis of the lower end of the esophagus: peptic ulcer. *a.* Roentgenogram of the esophagus showing herniation of the stomach with the firm cicatricial stricture at the junction of the stomach and esophagus. *b.* Roentgenogram of the stenosed area of the esophagus with a short (No. 30) cannulated bougie in position. Indwelling bougies No. 16, No. 18, No. 24 and finally No. 30 were introduced. They dilated the stricture until swallowing was normal. *c.* Roentgenogram showing a No. 44 mercury bougie passing through the strictured area of the esophagus into the stomach. Patient has gained 30 lbs. in weight on his normal diet.

BENIGN STENOSIS OF THE LOWER END OF THE ESOPHAGUS: PEPTIC ULCER

CASE 6.—L. K., male, 45 years of age. Admitted to the Graduate Hospital August 30, 1948, with a history of dysphagia and epigastric pain for the preceding three months. Esophagoscopy revealed infiltration of the lower end of the esophagus. Carcinoma was suspected but biopsy was negative. Roentgen examination revealed herniation of the stomach, narrowing at the junction of the esophagus and stomach. Mercury bouginage gave unsatisfactory results. Repeated esophagoscopies and biopsies were negative for malignancy. The patient was kept under observation for a year, refusing surgery. Finally a No. 16 indwelling bougie was introduced through the esophagoscope into the stenosed area and remained for 24 hours. Following this, the stricture dilated so that a No. 18, No. 20 and No. 24 bougie could be introduced. A No. 30 (Fig. *7b*) cannulated bougie remained in the esophagus for 12 hours and after this, mercury bougies were passed in increasing sizes until a No. 44 was admitted (Fig. *7c*). An esophagoscope was passed through the dilated stricture into the stomach and no evidence of ulceration or malignancy was found. Hurst mercury bougies were passed. Now the patient has no difficulty in swallowing, has gained 15 pounds in weight and food passes normally into the stomach. The result obtained with the indwelling cannulated bougie in this patient has been the most satisfactory in the group. The contemplated surgical resection of the lower end of the esophagus has been postponed indefinitely (Fig. *7a*, *b* and *c*).

FUNCTIONAL HIATAL STENOSIS. ACHALASIA

CASE 7.—Mrs. B. B., female, 28 years of age had a history of difficulty in swallowing for six and a half years, beginning with the patient's first pregnancy. On admission to the Graduate Hospital the patient was 5½ months pregnant and again was having serious trouble in swallowing. Esophagoscopy and treatment with mercury bouginage temporarily relieved her symptoms, but her general condition was such that gastrostomy was done. She was delivered of a normal child at term and was subsequently treated by mercury bouginage. A No. 30 indwelling bougie was placed in the hiatal esophagus for a period of 48 hours. Following this, two No. 30 short bougies were placed side by side in the hiatal esophagus for 24 hours. This afforded the patient satisfactory relief. Two weeks later a No. 40 cannulated bougie was introduced into the esophagus and permitted to stay 24 hours. The dysphagia was relieved for two weeks. This is our first experience with continuous dilatation of the hiatal esophagus with the indwelling bougie and further ob-

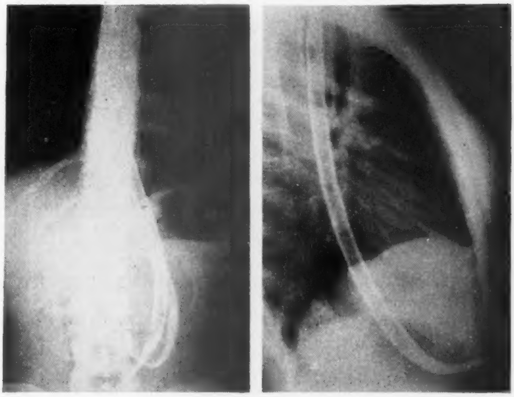

Fig. 8, Case 7.—Functional hiatal stenosis. Achalasia. *a.* Roentgenogram showing 2 short cannulated bougies (No. 30) in the hiatal esophagus. Patient swallowed fluids and purees normally with bougies in position. *b.* Roentgenogram showing size 40 cannulated bougie introduced through the hiatal esophagus. The bougie was introduced perorally through a previously swallowed string. The upper opening in the cannulated bougie is below the suprasternal notch, the lower opening is in the stomach. The bougie was suspended in this position by a string in the pharynx attached to an Equen "gadget" on an artificial denture.

servations will be reported. The peroral cannulated bougie can be placed in the hiatal esophagus and maintained in the proper position suspended by a string, through the nasopharynx. The patient can be given liquids and purees in as large quantities as desired (Fig. 8*a* and *b*).

CONCLUSIONS

This preliminary study of patients with stenosis of the esophagus (treated with the new Tucker indwelling cannulated bougie) has extended over a period of six months. Fourteen patients have been observed but not all are reported here.

Acute Lye Burns. Only one acute lye burn has been treated by this method; this one has responded very well to the indwelling bougie. It is believed that in this type of case the treatment period will be reduced to months instead of years as has been required by other methods of dilatation.

In the treatment of patients who had not responded to other types of treatment, the results with the new bougie have been most gratifying. Chronic strictures opened up almost immediately; and when the lumen of the esophagus was restored to normal, the patients were given daily or semi-weekly treatments by swallowing the Hurst mercury bougie of appropriate size. Observations have not been continued long enough to make certain of the end result, but in these cases the result to the present has been most encouraging.

Acute Peptic Ulcer. The use of the indwelling bougie for an acute peptic ulcer has been of temporary benefit and will be continued as cases present themselves. Results will be reported when sufficient study has been made to give a definite result.

FUNCTIONAL HIATAL STENOSIS AND CARDIOSPASM

In functional and hiatal stenosis and cardiospasm, we will be able, in those patients who do not have a gastrostomy, to use this method of continuous dilatation over a swallowed string (with the peroral type of indwelling bougie).

We hope to report further on this method of treatment with the Tucker indwelling bougie after the lapse of a year of observation and follow-up in all types of benign stenosis of the esophagus in all locations.

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LXXIX

BENIGN STENOSIS OF THE TRACHEA

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Benign stenosis of the trachea is described in most texts as usually luetic in origin. The fact that this has been only rarely encountered in bronchoscopic examinations during the past few years has prompted a review of this subject. A discussion of benign stenosis of the trachea requires a differentiation between those lesions of the tracheal wall which cause obstruction and extra-tracheal lesions which produce obstruction by compression.[6, 11] In this presentation the latter group is not considered.

This paper consists of a review of benign tracheal stenosis caused by lesions of the tracheal wall itself. It includes the congenital anomalies, specific and non-specific inflammatory lesions and stenoses of traumatic origin.

CONGENITAL ANOMALIES

Respiratory obstruction in infants presents a serious problem of diagnosis. In every case of chronic respiratory obstruction the tracheal anomalies must be considered.[15] In acute respiratory obstruction, particularly if there have been frequent recurrences of the obstruction with each upper respiratory infection, these anomalies must also be considered. Six types of congenital stenosis due to anomalies of the tracheal walls have been encountered in this series (Table I). The following case reports are examples of these anomalies:

K. N. (Case 1, Table I), white female, eight months of age, was admitted to The Children's Memorial Hospital for persistent croupy cough, stridor and cyanosis since birth. Delivery was normal. Examination showed a rough inspiratory stridor; harsh breath

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sounds were heard over the chest and both an inspiratory and an expiratory wheeze were noted, most pronounced on crying. The antero-posterior chest x-ray was negative, but the lateral film of the neck and chest revealed a tracheal stenosis below the level of the clavicles, the lumen of which was reduced at this point to approximately 1 mm. On bronchoscopic examination a marked tracheal narrowing with a horizontal slit-like stenosis was found at the level of the clavicles. The bronchoscope passed through this area easily, and the tracheal walls were found to be extremely flaccid. Collapse recurred as the bronchoscope was withdrawn.

This case was considered to be one of tracheomalacia and no treatment was indicated since obstruction was not sufficiently severe to require tracheotomy. Symptoms have gradually subsided and respirations are now clear.

M. H. (Case 4, Table I), white female, 34 months of age, was admitted to The Children's Memorial Hospital because of suprasternal retractions which had been noted for five days. Cough, dyspnea and cyanosis had been present for one day. She had had numerous upper respiratory infections with "croup," and during a severe upper respiratory infection which had been treated at another hospital five weeks previously it had been necessary to intubate her. Examination showed slight suprasternal retractions and a moderate inspiratory wheeze was heard over the entire chest. The lateral neck x-rays demonstrated a narrowing of the trachea below the glottis and a transverse band of increased density 3.5 cms below the glottis could be outlined. Tracheoscopy revealed a web below the conus elasticus with a small central perforation. This was incised and cautiously dilated several times during a period of one month. Respiratory obstruction was completely relieved and there have been no signs of recurrence.

The diagnosis of a congenital tracheal web had been suspected from the history and x-ray findings and was confirmed by the endoscopic examination.

M. N. (Case 5, Table I), white male, eleven months of age, was admitted to The Children's Memorial Hospital because of cyanosis and a slight stridor which increased during feedings. The symptoms had been present since birth and were accentuated by respiratory infections. At two months of age he had been bronchoscoped elsewhere, and a tracheotomy was done immediately following this. The tube was still in place on admission. Examination revealed many rhonchi over the entire chest, and x-rays demonstrated no air in the trachea between the tracheotomy tube and the larynx. Bronchoscopy revealed the tracheal walls from the larynx to a point beyond the tracheostomy to be extremely stenotic, with subglottic infiltration and no tendency to remain open after the bronchoscope was removed. The bronchoscope could be passed into both main bronchi which were normal. Treatment by dilatation was recommended and has been carried out elsewhere, the tracheotomy tube now having been removed.

This case represents a stenosis of a segment of trachea, presumed to be congenital in origin because of the findings of stenosis and the presence of symptoms since birth.

J. L. (Case 9, Table I), white male, four months of age, was admitted to The Children's Memorial Hospital because of a stridor which had been present since birth. Delivery was normal and without difficulty, but it was necessary that he be kept in an incubator for three weeks following birth because of respiratory difficulty. On two occasions of respiratory infections he was noted to have a persistent cough, dyspnea, dysphagia, cyanosis and sternal retractions. On examination he showed substernal and suprasternal retractions. Dullness was present over the right base and occasional inconstant rhonchi were heard over this area. X-rays showed a circular density interpreted as a tumor in the region of the right upper lobe which was anterior to the trachea. There was a hemivertebra of thoracic seven. Bronchoscopy revealed a marked tracheal stenosis immediately above what was thought to be the bifurcation, and the stenosis appeared to be due to compression from anteriorly. The right main bronchus could not be identified and the orifices of the left were anomalous. The infant was explored by Dr. Willis J. Potts, and an enlarged thymus found which accounted for the shadow on the chest film. A portion of this was removed but further exploration revealed a stenosis of the lower trachea; a blind stump of tracheal tissue was found pointing toward the right at a point thought to be the level of the bifurcation. The right bronchus apparently arose from the left main bronchus as illustrated in Fig. 1. The child had a somewhat stormy post-operative course but was discharged from the hospital about two weeks later. He continues to have noisy respirations but no further cyanosis or dyspnea.

This patient has a congenital stenosis of the distal trachea with an anomalous right bronchus.

B. T. (Case 10, Table I), white female, two weeks of age, was referred to The Children's Memorial Hospital with a history of cyanosis since shortly after birth. She had been placed in continuous oxygen for one week. Stridor, dyspnea, a wheeze and dysphagia

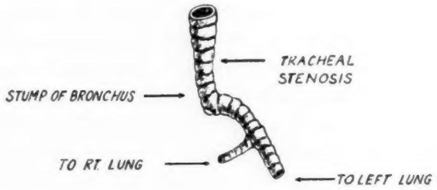
TRACHEAL STENOSIS
STUMP OF BRONCHUS
TO RT. LUNG
TO LEFT LUNG

Fig. 1.—(Case 9, Table II) Deformity of distal trachea showing stenosis, blind stump and apparent aberrant right bronchus.

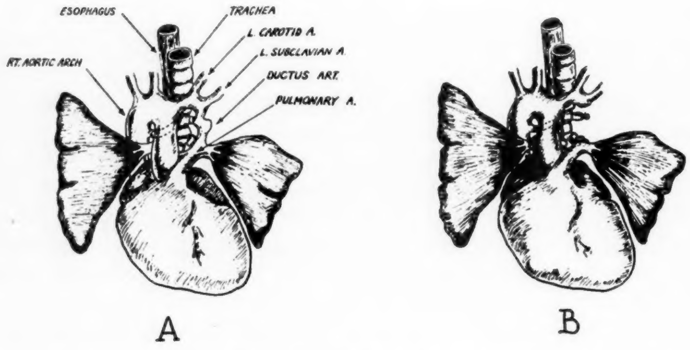
ESOPHAGUS
TRACHEA
L. CAROTID A.
L. SUBCLAVIAN A.
RT. AORTIC ARCH
DUCTUS ART.
PULMONARY A.
A
B

Fig. 2.—(Case 10, Table I) *A.* Vascular ring compressing trachea and esophagus. *B.* Persistence of tracheal stenosis following release of constriction by ligation of vessels.

had been noted during almost every feeding since birth. On examination there was a marked infrasternal retraction on inspiration, and rhonchi were heard throughout the chest. X-rays showed a definite narrowing and posterior indentation of the esophagus at the level of the arch of the aorta. A constriction of the trachea just above the bifurcation was also noted. Bronchoscopy revealed a constriction of the trachea about 1 cm above the caryna. On esophagoscopy there was a marked constriction at the same level which dilated easily, but extreme and almost fatal respiratory obstruction occurred when the esophagoscope passed through this esophageal stricture. These findings of both tracheal and esophageal constriction at the level of the aortic arch indicated a vascular ring. Four days after admission this patient was operated by Dr. Willis J. Potts. A vascular ring compressing the trachea and esophagus was found and the ring released as shown in Fig. 2. Respiratory obstruction was somewhat relieved, but twelve hours post-operatively a tracheotomy was necessary. This gave satisfactory temporary relief of the respiratory obstruction, but the infant had a stormy post-operative course with frequent recurrences of respiratory obstruction. It was determined that only a long tube reaching into either main bronchus would keep the airway open because of the persistence of the stenosis immediately above the coryna. A satisfactory airway could not be maintained because of ulceration and granulation tissue, and five weeks after operation a final fatal attack of obstruction occurred. Post-mortem examination showed a scarring and small ulceration of the right lateral wall of the trachea 1 cm above the caryna. The vascular ring, which had been severed, no longer compressed the trachea or esophagus.

This case demonstrates the effect of a right aortic arch with a vascular ring compressing the trachea and esophagus. Tracheal deformity persisted in spite of the satisfactory surgical release of the constricting ring.

An additional congenital anomaly in which stenosis of the trachea may ultimately develop is the congenital tracheo-esophageal fistula associated with esophageal atresia. Two infants present striking examples of this problem; the following is the case report of one of these infants:

T. J. (Case 11, Table I), white female, five and one-half months of age, was referred to the hospital because of persistent dysphagia and stridor following repair of a congenital esophageal atresia and a tracheo-esophageal fistula 30 hours after birth. These symptoms were present following operation and had increased when cereal was added to her diet. On examination many loud rhonchi were heard over the chest and a moderate respiratory stridor was noted especially with crying or feeding. X-rays exhibited a dilated one-third of the esophagus with only small traces of barium trickling through the site of anastomosis. Bronchoscopy revealed a definite narrowing of the trachea near the bifurcation through which the 3½ mm bronchoscope passed with difficulty only after several attempts at dilating the stenosis with the bronchoscope tip. There was a constriction of the tracheal wall at the site of repair of the congenital fistula into the posterior wall of the trachea. Esophagoscopy revealed a tortuous stricture through which the 3½ mm esophagoscope could not be passed. Following nine bronchoscopic dilatations in a period of four months the tracheal stenosis was markedly improved. This has permitted more vigorous dilatation of the esophageal stenosis and a good esophageal lumen has been re-established by retrograde dilatations following a gastrostomy.

In this infant who had a congenital esophageal atresia with a tracheo-esophageal fistula a constriction of the trachea was found at the level from which the distal segment of the esophagus was removed from the posterior wall of the trachea. This condition will be discussed in the section below.

DISCUSSION

A review of the literature demonstrates the fact that congenital stenosis of the trachea is not common. Montandon[17] divides the cases into the congenital fibrous strictures and stenoses produced by deformity or absence of the tracheal cartilages. Wolman[22] divides the stenoses into those of a uniform funnel-shaped constriction with the point of greatest narrowing immediately above the bifurcation, and a far less common hour-glass constriction of the upper portions of the trachea.

Twelve cases of congenital tracheal stenosis are reported in Table I with representative cases presented in detail above. Using the general classification of Montandon but subdivided according to the suggestions of Wolman and the findings in these cases they may be classified as follows:

- Congenital Fibrous Strictures
    - Webs—Hour-glass constriction (Case 4)
    - Fibrous stricture of a segment of trachea (Cases 5, 7)
    - Stenosis associated with congenital esophageal atresia and tracheo-esophageal fistula (Cases 11, 12)
- Stenosis due to absence or deformity of tracheal cartilages
    - Tracheomalacia (Cases 1, 2, 3)
    - Cartilaginous deformity (Cases 6, 8, 9)
    - Stenosis associated with vascular ring (Case 10)

Cases 4, 5, 7, 11 and 12 are classified as fibrous strictures. The infant described in Case 4 had a definite membranous web demonstrated on the lateral x-ray of the neck as being an hour-glass-shaped constriction of the trachea 3.5 cm below the level of the glottis. On inspecting this site with the bronchoscope the small, central opening through the web was seen. Careful dilatation gradually increased the size of the lumen so that a tracheotomy was unnecessary. The first of the fibrous strictures of a segment of the trachea seen in this series (Case 5) was examined after tracheotomy elsewhere had been necessary to relieve continuous chronic respiratory obstruction. The obstruction was present in the upper part of the trachea above the tracheotomy tube and gradually responded

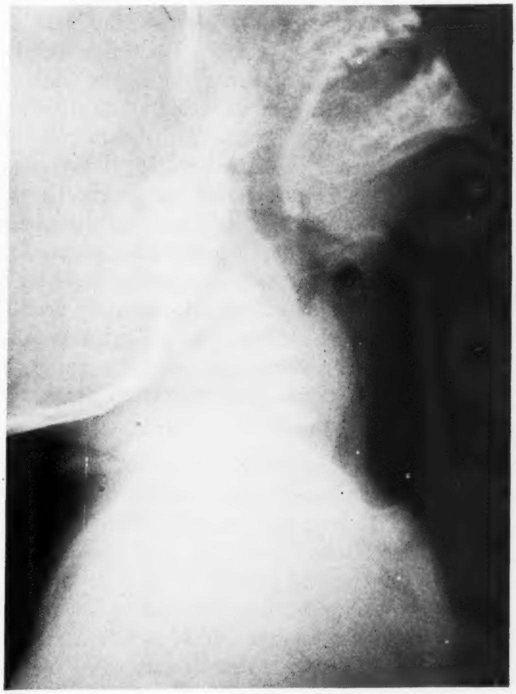

Fig. 3.—(Case 7, Table I) Lateral x-ray of soft tissue of the neck showing congenital stricture of a segment of the trachea.

to dilatation. The stenosis did not involve the larynx itself but began below the level of the cricoid cartilage. The second case listed in this category (Case 7) is included here although no endoscopic examination was made. The infant had an inspiratory and expiratory stridor and x-rays demonstrated a tracheal stenosis which persisted during both phases of respiration (Fig. 3). No endoscopic procedure was made because of the fear of precipitating a tracheotomy and, as the infant gradually improved, further work was deemed unnecessary.

Two cases of congenital esophageal atresia with tracheoesophageal fistula are included in this series (Cases 11, 12) because of the finding of a fibrous stricture in the trachea. Both infants had a stormy post-operative course following the original operation in the newborn period at the time of the anastomosis of the upper and lower segments. Their respiratory obstruction was a prominent feature of the post-operative period and continued to be a major problem during their first two years. Each developed a stenosis at the point of the anastamosis between the upper and lower esophageal segments. Their dyspnea and wheezing made attempts at esophageal dilatation hazardous for several months because of extreme cyanosis during the procedure. Consequently, bronchoscopic examinations were made which demonstrated the tracheal stenosis at the point from which the lower esophageal segment had been removed from the trachea and the tracheal dehiscence closed. It is difficult to determine whether these are true congenital tracheal stenoses or whether they are the result of cicatricial contraction of the trachea at the point of the surgical closure. In 16 other infants who are under observation following surgical repair of a congenital esophageal atresia with tracheo-esophageal fistula no tracheal stenosis is apparent.

Seven cases of congenital tracheal stenosis are considered in the group of stenosis due to absence or deformity of tracheal cartilages. Three infants (Cases 1, 2 and 3) demonstrated the definite characteristics of tracheomalacia. In presenting these cases under the heading of congenital tracheal stenosis it is necessary to point out that the tracheal narrowing was not sufficiently severe to cause fatal termination. Nevertheless, symptoms of severe stridor with associated cyanosis and dyspnea were manifestations of marked obstruction. In each instance the tracheal wall was exceptionally flaccid. The disappearance of symptoms with the passage of the bronchoscope through the narrowing and the fact that symptoms disappeared with increasing age and the bronchoscopic evidence of the flaccid tracheal wall indicated the presence of a localized retarded development of the tracheal wall. The flaccidity was much more pronounced than that seen in the allergic infant and no evidence of allergy was present in these three infants. Lateral x-rays of the neck and chest demonstrated the stenosis visible on bronchoscopy.

Cartilaginous deformity as a cause of tracheal stenosis was present in three cases of this series (Cases 6, 8, 9). Numerous cases were reviewed in which a deformity of the subglottic region was noted, but careful analysis demonstrated them to be due to abnormalities of the cricoid cartilage and, consequently, these were not included in the present discussion. The stenoses in cases 6 and 8 were found at the level of the first tracheal ring, below the cricoid. They were visible on x-ray and endoscopically were found to be cartilaginous in rigidity. The openings were posteriorly. The third case of this subgroup (Case 9) was described in detail above and consisted of a funnel-shaped stenosis of the distal trachea. In this infant an unusual bronchial anomaly was associated with the tracheal deformity. The character of the stenosis of the distal trachea suggests that the deformity in this infant may be classified in the first group suggested by Wolman, namely, a funnel-shaped constriction with the point of greatest narrowing at the distal end of the trachea.

Congenital anomalies of the cardiovascular system producing tracheal stenosis are now well recognized. Case 10 is illustrative of this anomaly. A previous report on this subject summarizes the cases that fall into this category.[9] Five infants and children were presented in whom congenital anomalies of the aorta and its main branches produced severe tracheal stenosis as the predominating symptom. Three additional infants have been seen in whom tracheal stenosis has been found to be caused by vascular constriction. Detailed case histories of these three additional cases are essentially the same as those presented in the previous publication. The symptoms are progressive dyspnea, generally reaching alarming proportions at four to five months of age, with dysphagia and cyanosis associated with feedings. In Case 10 of this group, the dyspnea and dysphagia were present from birth. The significant finding in these infants has been the persistent deformity or absence of tracheal cartilages at the site of the vascular ring as illustrated in Fig. 2. This was demonstrated, too, in Case 7 of the previous report. Thus, the constriction is apparently not alone a compression stenosis but is, in fact, an actual persistent deformity of the tracheal cartilages.

ACQUIRED TRACHEAL STENOSES

Acquired tracheal stenoses due to lesions of the tracheal wall may be either inflammatory or traumatic in origin.[1, 2] A larger group of cases cause tracheal stenosis by extrinsic pressure but, as mentioned above, these are not included in this discussion.

Inflammatory. Six cases of non-specific inflammatory tracheal stenosis are found in this series, one of which is presented in detail.

M. H. (Case 16, Table II), white female, 44 years of age, was admitted to The Illinois Research and Educational Hospitals for an acute attack of "asthma" which had persisted for three days. For about seven months she had had a whooping type of cough and wheeze had been present for the past four months. Examination showed a marked dyspnea. Inspiratory crowing respirations and marked expiratory rhonchi were heard over the right lower chest. Repeated Kahn and Wasserman tests were strongly positive. On bronchoscopic examination there was a concentric stenosis of the trachea beginning about 2 cm above the caryna with both main bronchial orifices markedly constricted. There was no response to bronchoscopic dilatation. The patient later developed a severe bronchopneumonia which did not respond to sulfa therapy and died. Post-mortem examination revealed a marked stenosis of the lower trachea. The lumen of the right bronchus was 2 mm and the left bronchus 5 mm in diameter. Microscopically the pathology was that of a chronic fibrotic inflammation of the tracheal and bronchial walls. There was no microscopic evidence of a syphilitic or tuberculous disease process.

This case is considered to be a non-specific inflammatory tracheal stenosis on the basis of the histologic findings.

In two of the patients of this group (Cases 13, 14, Table II) the inflammatory process involved the nose, pharynx, larynx, and trachea. In each of these patients rhinoscleroma was strongly suspected but never could be proved by biopsy or culture. A third patient, a child (Case 15, Table II), had an extensive process involving the pharynx, larynx, trachea, and esophagus. Scar tissue eventually formed tight stenoses of these structures, thus differing from the soft inflammatory tissue obstruction in the first two cases. The fourth patient in this group, reported in detail above (Case 16, Table II) had a stenosis of the distal trachea without any associated laryngeal pathology. Histologically the stenosis proved to be a thickening of the tracheal and bronchial walls from a chronic inflammatory process. There was no microscopic evidence of syphilis or tuberculosis although the patient's serology was repeatedly positive. The fifth patient had severe chronic pemphigus with extensive pharyngeal, laryngeal and tracheal stenosis requiring tracheotomy and gastrostomy.

One unusual web-like tracheal stenosis is included in this category since no specific inflammatory process could be identified as its etiologic agent:

N. G. (Case 18, Table II), white female, 57 years of age, stated she had noticed a wheeze in her chest for about eight years. There was an associated slowly progressive dyspnea and occasional hoarseness. No history of trauma, tuberculosis, or severe acute respiratory tract infection could be obtained. Serology was negative. On examination a soft inspiratory and expiratory stridor was heard. A postero-anterior film of the chest was negative for evidence of tuberculosis, but x-rays demonstrated narrowing of the trachea at C-7; planographic studies confirmed this at the levels of 9, 10 and 11 cm. Bronchoscopy revealed a marked constriction of the tracheal lumen 3.5 cm below the vocal cords; .5 cm below this narrowing was a

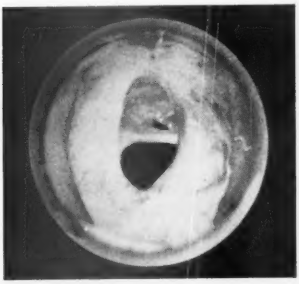

Fig. 4.—(Case 18, Table II) Endoscopic photograph of web-stenosis of the upper trachea. Two levels of webbing are shown, with a small and larger opening in the lower web.

similar constriction with a membranous web across the anterior trachea (Fig. 4). After two bronchoscopic dilatations the lumen was restored to normal.

This patient represents a web-like tracheal stenosis of unknown etiology.

Specific inflammatory diseases of the trachea causing stenosis include syphilis,[7, 13] tuberculosis, diphtheria, rhinoscleroma, and typhoid fever. The following are representative of this group.

V. H. (Case 19, Table III), white female, 36 years of age, was admitted to St. Luke's Hospital because of a slowly increasing swelling in the midline of her neck over a period of five months. She complained of dyspnea for two months and continual hoarseness for three weeks. On examination there was a firm, movable, golf-ball sized mass in the midline below the larynx. Her voice was hoarse. Both Wasserman and Kahn serologies were strongly positive. On direct laryngoscopy the left arytenoid was edematous. Bronchoscopy revealed a soft granulomatous mass protruding from the upper posterior tracheal wall. Biopsy of this tissue demonstrated chronic gummatous inflammation. Following anti-luetic therapy her symptoms have regressed.

Serology and biopsy in this case proved the tracheal stenosis to be produced by a gummatous infiltration of the tracheal wall.

L. B. (Case 20, Table II), white female, 54 years of age, entered St. Luke's Hospital complaining of cough and exertional dyspnea for 48 years. In the past two weeks she had noted a persistent hemoptysis. Hoarseness and a wheeze followed intubation for diphtheria at the age of six. Examination showed slight cyanosis and dyspnea. Coarse rales and rhonchi were heard over the right chest. Bronchograms demonstrated saccular bronchiectasis of the right lower lobe and fusiform bronchiectasis of the left lower lobe. Laryngoscopy exhibited fibrosis with marked deformity of the larynx. Bronchoscopy revealed a marked stenosis 2 cm below the larynx which did not allow passage of the 5 mm bronchoscope. A tracheotomy was done and repeated laryngeal bouginage and bronchoscopic dilatations over a year produced an adequate tracheal lumen.

A post-diphtheritic laryngeal and tracheal stenosis is presented by this case.

A. M. (Case 24, Table III), female Japanese, 26 years of age, had noted severe dyspnea, cyanosis and cough following a right pneumothorax refill two days before admission to St. Luke's Hospital. Pneumothorax had been initiated five months prior to this, one month after her initial symptoms of tuberculosis. On examination breath sounds were faint to absent over the left chest but loud rhonchi were heard over the trachea. The typical findings of pneumothorax were present on the right. There was marked tachycardia, dyspnea, and cyanosis. Her sputum was repeatedly positive for acid-fast bacilli. X-rays demonstrated a right pneumothorax with atelectasis of the right middle and lower lobes. The trachea was displaced to the left and the lumen indistinct. Bronchoscopy revealed the posterior part of the larynx to be thickened. The trachea was filled with caseous, purulent material. After aspiration the trachea was found to be extremely narrow and the entire right tracheal wall was seen to be replaced by soft polypoid granulomatous tissue producing a 50% stenosis. The right bronchus was occluded by caseous granulation tissue. The patient developed cardiac decompensation and died five days later.

This patient had extensive tuberculous stenosis of the entire trachea with a superimposed acute ulcerative caseous tuberculous tracheitis.

According to the older textbooks it is apparent that syphilis was the commonest cause of benign tracheal stenosis.[14] Negus[21] indicates that this condition has ceased to be a leading factor at the present time, and in our series of cases only one luetic stricture is reported. This does not include cases of tracheal compression due to aneurysms, of which numerous cases were encountered.

It is reasonable to suspect that tracheal stenosis due to diphtheria and typhoid fever will be extremely rare in this country because of the constantly decreasing incidence of these disease. Tracheal stenosis due to tuberculosis is not infrequent and only one case is included in this report. The recognition of this complication of pulmonary tuberculosis and the more widespread use of bronchoscopic examination in suspected cases will result in its earlier detection and thus in the early institution of appropriate surgical and chemotherapeutic measures to combat it.

Rhinoscleroma with tracheal stenosis presents one of the most serious problems for the laryngologist in other countries, notably Mexico, where many patients with tracheal stenosis due to this disease are seen. Reports of similar cases in this country are appearing in the literature.[5] It must be considered in all chronic inflammatory lesions infecting the upper and lower respiratory tracts but the diagnosis is sometimes difficult to establish. The two patients originally considered to have rhinoscleroma in this series (Cases 13 and 14, Table II) did not have the clinical criteria necessary to consider this diagnosis without some question.

Traumatic. Five cases of traumatic stricture of the trachea are presented. In three of the cases a traumatic fracture of the trachea was present; in two of these the intrathoracic portion of the trachea was fractured in automobile accidents. In the third case the cervical portion of the trachea was involved. This patient was struck in the neck by a golf ball which fractured three tracheal cartilages, requiring tracheotomy because of obstruction. The fourth case of traumatic stricture of the trachea followed a thyroidectomy; while in the fifth case a moderate stenosis persisted at the point of a tracheotomy. The following case is representative of the stenosis following fracture which occurred in an automobile accident.

O. D. (Case 26, Table IV), white male, 62 years of age, entered St. Luke's Hospital because of persistent hoarseness of over four months. The patient had sustained fractures of the right scapula, the sternum, and ribs on both sides of the sternum in an automobile accident three years previously. His condition at that time was complicated by a right pneumothorax. Physical examination was essentially negative except for the laryngeal pathology. The serology was negative. Direct laryngoscopy showed a granular, ulcerating lesion involving the left vocal cord and ventricle. Biopsy proved this to be squamous cell carcinoma, and surgical removal of the larynx was advised. At the time of the laryngectomy difficulty was encountered in inserting the endotracheal tube after removal of the larynx. A stenosis of the trachea was found about 5 cm below the stoma allowing only a No. 5 cane-shaped tracheotomy tube to be inserted. A subsequent bronchoscopic examination made through the tracheal stoma demonstrated the marked stenosis at 5 cm to be

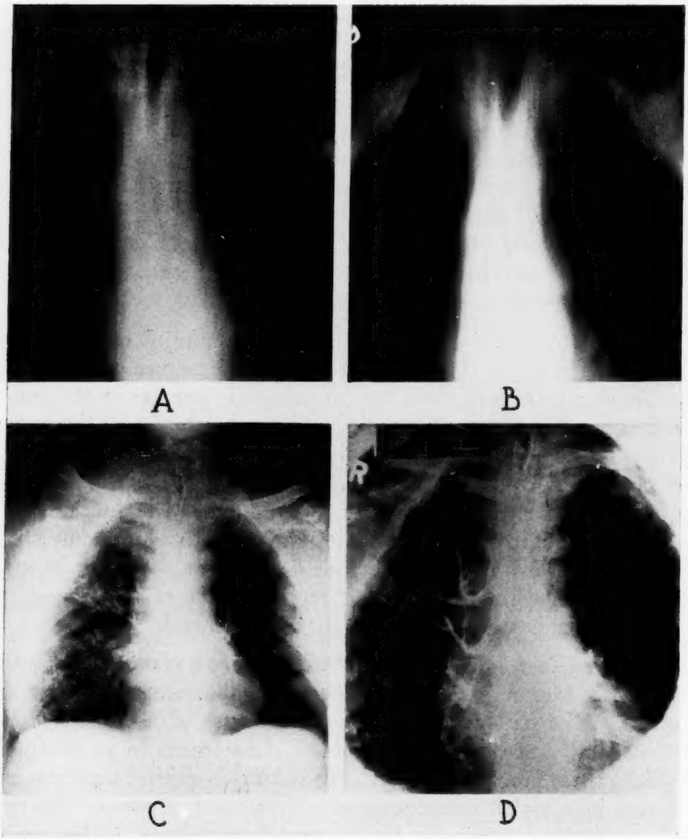

Fig. 5.—(Case 26, Table IV) Traumatic tracheal stenosis and congenital anomaly of the right upper lobe bronchus. *A.* Planogram demonstrating aberrant right upper lobe bronchus. *B.* Planogram showing tracheal stenosis. *C* and *D.* Bronchograms demonstrating the degree of stenosis and the position of the right upper lobe bronchus.

smooth and concentric. At 10 cm an accessory right upper lobe bronchial orifice was seen. The coryna was found to be 2 cm below this accessory bronchus. Figure 5 shows a bronchogram made at a later date. The accessory bronchus is seen originating on the right wall of the trachea. It appears to aerate a segment of lung corresponding to the apical segment of the right upper lobe.

This patient had a tracheal stenosis following an automobile accident in which multiple fractures of the thoracic cage and sternum occurred and during which a pneumothorax developed. Carcinoma of the larynx and an anomalous right upper lobe bronchus were unrelated findings.

D. Z. (Case 27, Table IV), white female, 6 years of age, was accidentally struck in the neck by a golf ball. She immediately developed severe pain and extreme dyspnea. Examination showed a golf ball imprint in the midline of the neck anteriorly, and brawny bluish swelling just below the cricoid area. X-rays demonstrated slight anterior tracheal deviation with retropharyngeal air present. Prior to a tracheotomy, direct laryngoscopy and bronchoscopy revealed a hematoma and edema of the larynx with minimal laryngeal obstruction. There was a tracheal compression due to fractures of the second, third and fourth tracheal cartilages, portions of which were seen to enter the tracheal lumen. A low tracheotomy was done with immediate relief of symptoms. She was extubated in approximately six weeks and has remained asymptomatic following extubation.

This patient sustained three fractured tracheal cartilages when a golf ball struck her in the neck. Acute tracheal stenosis developed due to the fractured tracheal cartilages which protruded into the tracheal lumen.

J. W. (Case 28, Table IV), white female, 41 years of age, noted increasing cough and dyspnea immediately following a thyroidectomy. Bronchoscopy revealed a normal motility of the vocal cords on inspiration and phonation, but a stenosis of the trachea was observed at the level of the thyroidectomy scar. Bronchoscopic dilatations rapidly relieved her symptoms and eventually breathing improved sufficiently to permit extubation.

This patient had a tracheal stenosis in the region of the thyroid scar following thyroidectomy.

M. L. (Case 29, Table IV), white male, eight months of age, required an emergency tracheotomy for acute laryngotracheobronchitis two months before admission to The Children's Memorial

Hospital. There was no history of previous respiratory obstruction. All attempts at extubation following this acute process had failed. On examination rhonchi were heard throughout both lungs due to tracheal mucus accentuated by the presence of the tracheotomy tube. Bronchoscopy revealed a narrowing of the trachea at the level of the tracheotomy stoma with no stenosis above or below this point. Bronchoscopic dilatations of the stenosis allowed extubation after four weeks' treatment. The patient had another attack of laryngotracheobronchitis 14 months later which required only medical management.

This case represents a tracheal stenosis following a tracheotomy which responded to bronchoscopic dilatation.

The cases of traumatic stenosis of the trachea are of unusual interest. Clerf[4] has shown that x-ray can be an additional etiological agent in this group. Stenoses following cut-throat or gun-shot wounds of the trachea above the clavicles are evident[16] and often surgical repair with skin grafting is necessary. The emergency treatment usually consists of a tracheotomy, but prompt endoscopic manipulation of the fractured ends, after the initial shock has been treated, replacing them as accurately as possible may hasten healing and restoration of the normal lumen. The insertion of an acrylic mold or polyethylene tube above a low tracheotomy to maintain the airway during the healing process is of benefit.[19]

Fracture of the trachea occurring inside the thoracic cage presents an entirely different problem. The two cases herein reported were seen after stenosis developed. Similar cases of fracture of the bronchus have been reported previously,[10] with complete atresia of a major bronchus resulting from automobile accident injury. It would appear from an analysis of the cases of fracture of the trachea and from those previously reported of fracture of the bronchus that inspection of the tracheobronchial tree is indicated if a pneumothorax is an associated finding in a severe injury. Early dilatation of a stenosing trachea or bronchus may reduce the time required for treatment or may maintain the patency of a bronchus which would otherwise become completely and irreversibly obstructed.

Analysis of this entire series of benign stenoses of the trachea demonstrates that each patient presented the well known symptoms of tracheal obstruction, namely wheezing, stridor, croupy cough and, in severe cases, marked dyspnea and cyanosis. The absence of voice changes and the demonstration of bilateral pulmonary findings indicated the general location of the obstruction to be in the trachea. Of great assistance in the establishment of the diagnosis was the roentgen study which included the lateral x-ray of the neck and the lateral chest x-ray.[12] In infants this must be made with the arms down and back, the neck forward and the head partially extended. Planograms in both the antero-posterior and the lateral planes are of great assistance in some instances. The final diagnosis rests upon the endoscopic examination with the removal of tissue, if possible, for histologic study.

SUMMARY

1. Twenty-nine cases of benign tracheal stenosis due to lesions of the tracheal wall itself are presented. Twelve are congenital in origin; seventeen are due to acquired lesions.

2. The congenital anomalies producing stenosis are divided into the congenital fibrous strictures and those due to absence or deformity of the tracheal cartilages. Webs, fibrous strictures of segments of the trachea and stenoses associated with congenital esophageal atresia and tracheo-esophageal fistula are included in the first group. Tracheomalacia, cartilaginous deformities and persisting deformity stenoses associated with the vascular rings are included in the second group.

3. Of the acquired tracheal stenoses, six were due to nonspecific inflammatory processes.

4. The specific inflammatory diseases considered as etiologic factors in tracheal stenosis are syphilis, tuberculosis, diphtheria, typhoid fever, and rhinoscleroma. Six representative cases are presented in this group.

5. Five cases of stricture of the trachea of traumatic origin are discussed. In two, fractures of the thoracic portion of the trachea occurred in automobile accidents. Two were postsurgical strictures of the cervical trachea, and one was a golf ball fracture of the cervical trachea.

6. The diagnosis of benign tracheal stenosis is suspected from the history and physical findings. Careful x-ray study and the endoscopic examination are essential in establishing the final diagnosis.

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TABLE 1.—SUMMARY OF CASES OF CONGENITAL TRACHEAL STENOSIS.

No.	Name	Age Sex Race*	Symptoms	Physical Examination	X-rays and Laboratory	Endoscopic Findings	Treatment	Final Result	Final Diagnosis
1	KN	8 mo. F	cough, stridor, cyanosis	inspir. & expir. stridor; wheeze	stenosis below clav. lumen 1 mm.	Slit stenosis at clavicles. Flaccid tracheal walls	none	good	Tracheomalacia
2	PP	3 mo. F	cough, stridor, dyspnea, wheeze	inspir. stridor	stenosis at clav.	ant. post. stenosis above coryna, dilates easily	none	good	Tracheomalacia
3	JH	8 mo. F	stridor, dyspnea, cyanosis	stridor, sternal retractions	narrowing at clav. lumen 2 mm	soft narrowing in mid trachea, dilated easily	none	good	Tracheomalacia
4	MH	34 mo. F	retract., cough, dyspnea, cyanosis	retractions, inspir. wheeze	stenosis 3.5 cm below glottis	web with cent. perforation below conus elasticus	incision and laryngeal dilatations	complete relief of obstructive symptoms	Congenital tracheal web
5	MN	11 mo. M	stridor, cyanosis	rhonchi tracheotomy tube	air absent above tracheotomy tube	lumen stenotic, walls infiltrated,	tracheotomy, dilation of stenosis	improved; tracheotomy tube removed	Congenital segmental tracheal stenosis
6	BS	27 mo. F	cough, dyspnea	hoarseness, rhonchi	stenosis 1 cm below cords, 3 mm lumen	stenosis of ant. 1/3, 1 cm below cords	dilations over 3 mo.	improved	Congenital tracheal stenosis
7	PR	4 mo. F	dyspnea, stridor, cough, wheeze	sternal retract. hoarse cry	5 cm stenosis at C3-4 level	no procedure	none	unchanged	Congenital tracheal stenosis
8	RI	18 mo. M	cough, wheeze, stridor	expir. wheeze, sternal retract.	2 cm stenosis below glottis	firm stenosis below cricoid	none	unchanged	Congenital tracheal stenosis
9	JL	4 mo. M	stridor, dysphagia, dyspnea, cyanosis	sternal retract. occas. rhonchi	density over rt. upper lobe	stenosis above bifurcation. Anomaly rt. bronchus	thoracotomy	improved	Congenital tracheal sten. with anomalous rt. bronchus

TABLE 1.—Continued.

No.	Name	Age Sex Race*	Symptoms	Physical Examination	X-rays and Laboratory	Endoscopic Findings	Treatment	Final Result	Final Diagnosis
10	BT	2 wks. F	cyanosis, wheeze, stridor, dysphagia	retract. rhonchi tracheal wheeze	stenosis of trach. & esoph. at aortic arch	stenosis of trach. & esoph. 1 cm above coryna	Surgical release of vascular ring. Tracheotomy	died	Rt. aortic arch with vascular ring. Tracheal deformity
11	TJ	5½ mo. F	dysphagia, stridor	stridor; rhonchi. Surgical repair of cong. esoph. atresia	esoph. strict. at anastomosis	tracheal stenosis near coryna. Esoph. strict. at anast. site	tracheal dilations over 4 mo. Esoph. strict. under treatment	improved persistent esoph. strict.	Tracheo-esoph. fistula with tracheal stenosis
12	RQ	2 mo. M	regurg. dysphagia, dyspnea	Repaired esoph. atresia. Wheeze, retractions.	Trach. stenosis, esoph. stenosis at point of anastamosis.	Tracheol sten. near coryna. Slight esoph. stricture	Tracheal dilations. Esoph. dil.	improved minimal esoph. strict.	Tracheo-esoph. fistula with tracheal stenosis

*All infants in this Table, white race.

TABLE 2.—SUMMARY OF CASES OF NON-SPECIFIC INFLAMMATORY TRACHEAL STENOSIS.

No.	Name	Age Sex Race*	Symptoms	Physical Examination	X-ray and Laboratory	Endoscopic Findings	Treatment	Final Result	Final Diagnosis
13	DP	28 yr. F	cough, wt. loss, dyspnea	rasping breath sounds lt. base, scarred nasal septum, pharynx & larynx	tracheal narrowing at level of C-6	soft laryngeal stenosis, continuing in upper trachea; dilated to admit 6 mm. scope	dilations over 6 mo. tracheotomy	improved and extubated	Non-specific inflam. tracheal and laryngeal stenosis ,biopsy diagnosis)
14	AK	57 yr. F	stridor, dyspnea, hoarseness	sternal retract., scarred pharynx, nasoph, larynx, trachea	narrowing of upper trachea	scarred larynx, inflam. tissue narrowing trachea to 3 cm. below glottis	tracheotomy & dilations over 21 mo.	improved and extubated	Non-specific inflam. laryngea & trach. stenosis (biopsy diagnosis)
15	VP	1 mo. M	cough, dyspnea, hoarseness, fever	dyspnea, stridor, coarse rales and rhonchi in chest	stenosis 3 cm. below glottis, inc. retropharyngeal tissue, chest film negative	edematous, scarred epiglottis larynx & esoph. Stenosis upper trach.	tracheotomy & dilations over 33 mo. Gastrostomy	improved	Non-specific esoph. laryng. & trach. stenosis
16	MH	44 yr. F	cough, wheeze, dyspnea	dyspnea, expir. crowing respir. & rhonchi	chest film negative. Serology positive.	Concentric stenosis 2 cm. above coryna & both main bronchi narrowed	dilations chemotherapy	died	Non-specific tracheal stenosiis (Histologic diagnosis)
17	MN	64 yr. F	increasing dyspnea & dysphagia	rales & wheezes over chest. Scarring & ulcers of tongue, palate, pharynx	diffuse pneumonitis in both bases	extensive scarring of pharynx, larynx with tracheal stenosis	gastrostomy & tracheotomy	uninproved	Chronic pemphigus with pharyngeal, laryngeal & tracheal stenosis
18	NG	57 yr. F	progressive dyspnea 8 yrs.	soft inspir. expir. stridor	narrowing at level of C-7	Thin membrane 2.5 cm below cricoid with 2 small openings	dilations twice	improved airway normal	Web-like tracheal stenosis

*All patients in this Table, white race.

TABLE 3.—SUMMARY OF CASES OF SPECIFIC INFLAMMATORY TRACHEAL STENOSIS.

No.	Name	Age Sex Race*	Symptoms	Physical Examination	X-ray and Laboratory	Endoscopic Findings	Treatment	Final Result	Final Diagnosis
19	VH	36 yr. F	swelling in neck, dyspnea, hoarseness	movable midline mass below larynx; hoarseness	essentially negative chest & neck x-ray. Serology positive	lt. arytenoid edematous; swelling of post. tracheal wall	anti-luetic therapy	improved	Gummatous tracheal stenosis (Biopsy diagnosis)
20	LB	54 yr. F	hoarseness hemoptysis wheeze. Diphtheria & intubation at 6 yrs. of age.	slight cyanosis & wheeze, rales & rhonchi over rt. chest	bronchograms: saccular bronchiect. RLL, fusiform of LLL	fibrotic deformed larynx, stenosis 2 cm below larynx; unable to pass 5 mm scope	tracheotomy & dilations over 1 yr.	improved & extubated	Post-diphtheritic laryngeal & tracheal stenosis
21	IC	22 yr. F	hoarseness & wt. loss. Diphtheria & intubation at 2 yrs. of age	tricuspid heart murmur	lat. chest & neck studies ess. neg.	larynx deformed & stenotic; 3 cm below larynx trachea stenotic and scarred	tracheal dilations	improved	Post-diphtheritic laryng. & trach. stenosis
22	MN	46 yr. F	Increas. cough & dyspnea, Tbc. 20 yrs.	trach. deviated to rt. flatness & decreased breath sounds over rt. chest	rt. chest dense, cavity at D-6, emphysema on lt. sputum pos.	"corkscrew" stenosis from larynx to coryna; both bronchi narrowed, rt. pinpoint	none other than supportive	uninproved	Tuberculous tracheal & bronchial stenosis
23	AT	58 yr. M	cough & dyspnea, cyanosis, retract. Tbc. 15 yrs.	inspir. wheeze & stridor, rales bilat. cyanotic	stenosis at D-2 bilat. multiple cavities sputum pos.	Stenosis 3 cm below cords, mucosa red, lumen 6 mm	dilations & tracheotomy over 30 mo.	died	Tuberculous tracheal stenosis
24	AM	26 yr. F	severe dyspnea, cough & cyanosis Tbc. 5 mo.	absent breath sounds on rt., inspir. expir. rales over lt. chest	rt. pneumo. with atelect. of middle & lower lobes. Tracheal lumen indistinct. Sputum positive	post. larynx thick., rt. trach. wall replaced by granulations, 50% stenotic	aspirations & supportive	died	Tuberculous tracheal stenosis

*Cases No. 19-23, white. Case No. 24, yellow.

TABLE 4.—SUMMARY OF CASES OF TRAUMATIC TRACHEAL STENOSIS.

No.	Name	Age Sex Race*	Symptoms	Physical Examination	X-ray and Laboratory	Endoscopic Findings	Treatment	Final Result	Final Diagnosis
25	JE	33 yr. M	dyspnea, pneumothorax following auto accident. Tracheotomy	respiratory obstruction in spite of tracheotomy	stenosis distal trachea	stenosis 4 cm above coryna, dilated to 7 mm scope	dilatations with bronchoscope and special trach. tube	improved tracheotomy tube removed	Post-fracture tracheal stenosis
26	OD	62 yr. M	persist. hoarseness; history of auto accident with sternum and rib fractures	tumor left vocal cord (biopsy; ca.)	sten. 7 cm above coryna accessory RUL bronch. 2 cm above coryna	stenosis 7 cm above coryna, access. RUL bronch. 2 cm above coryna	Laryngectomy. Cane-shaped laryngect. tube	improved tracheal stenosis dilated with cane tube	Post-fracture tracheal stenosis; ca. of larynx
27	DZ	6 yr. F	marked dyspnea & pain over cervical trachea	bluish swelling below cricoid	tracheal compression retro-pharyngeal air	hematoma of larynx, fract. 2, 3, 4 rings seen within trach. lumen	tracheotomy	good extubated in 3 weeks	Post-fracture tracheal stenosis
28	JW	41 yr. F	cough & dyspnea post thyroidectomy	acute resp. distress	none	stenosis of trachea at level of thyroid scar	dilations over 3 yrs. tracheotomy	good	Post-thyroidectomy. Tracheal stenosis
29	ML	8 mo. M	dyspnea on attempted extubation	rhonchi over entire chest	none	stenosis level of tracheotomy stoma	dilations over 2 weeks	good extubated	Post-tracheostomy. Tracheal stenosis

*All patients in this Table white race.

Abstracts of Current Articles

EAR

Rhinosporidium In Iran.

Kossar, M.: Les annales d'oto-laryngologie 66:470-476 (Sept.) 1949.

The lesion, occurring within the nasal fossa, is a projecting polypoid mass of tissue, having a rough, granular, bleeding surface. Microscopically, one finds numerous young spores and mature sporangia in the epithelial layer. The epithelium is hyperplastic and shows desquamation and superficial ulceration. In the tunica propria, one finds the parasites in all stages of development. Giant cells are present, many showing phagocytosis of parasites. There are surrounding rings of polymorphonuclear leucocytes, intense vascular congestion and interstitial hemorrhages, infiltration with plasma cells, lymphocytes and eosinophiles.

Clinically, the condition is characterized by epistaxis, nasal obstruction and pain. Usually it is unilateral. The affected mass of tissue varies in size from a grain to that of a hazelnut. It may present itself on the exterior of the nose. The disease may attack the external structures of the eye. The method of transmission is unknown; the spores are present in the nasal secretions and the tears.

Four cases' histories are presented. Emetine has been proposed for treatment; it did not prove successful among the Iranian cases. The most satisfactory treatment seems to be surgical excision of the involved tissue, followed by electrocoagulation of the base. Recurrence of the infection is common.

The disease has been reported in horses in South Africa and the East Indies. Attempts to transmit the human infection to rabbits, sheep and calves have not been successful.

HARKINS.

Streptomycin Treatment in Tuberculous Otitis Media.

Siirala, V., and Lahikainen, E. A.: Arch. Oto-Laryn. 39:6-528 (Dec.) 1949.

Five cases of proven tuberculous otitis media are reported in which surgical and streptomycin treatment resulted in healing. Three cases developed suppurative fistulae, two in the postoperative scar and one which came on preoperatively. Following streptomycin therapy the fistulae closed and the ears became dry.

HILL.

Concurrence of Glaucoma and Meniere's Disease.

Godtfredson, E.: Arch. Oto-laryn. 37:6-533 (Dec.) 1949.

The parallelism between the paroxysms in Meniere's Disease and increases in intraocular pressure suggests a common angioneurotic pathogenic factor. Three cases are presented in detail which showed concurrent glaucoma and labyrinthine hydrops. Possibly similar cases may be overlooked due to the attention necessarily devoted to the ocular condition.

HILL.

Plastic Correction of Congenital Atresia of the External Auditory Meatus Combined with Fenestration to Improve Hearing.

Sürala, V.: Arch. Oto-laryn. 37:4-307 (Aug.) 1949.

The author reports a case of congenital meatal atresia with microtia, in which plastic operation on the external meatus was followed by fenestration. A rectangular skin flap was raised behind the auricle and after mastoidectomy carried into the cavity by means of an endural incision, thus forming the posterior wall of the new canal. The malleus and incus were removed and the horizontal semicircular canal fenestrated. A Tiersch skin graft was used to cover the fenestra and the operative cavity. Improvement in hearing was noted. Inasmuch as the hearing in the other ear was normal one might question the indications for fenestration in this case.

HILL.

Tympanic Body Tumors in the Middle Ear—Tumors of Carotid Body Type.

Lundgren, N.: Arch. Oto-laryn. 37:4-367 (Aug. 1949.

The author reports four cases of his own and discusses nine other cases reported in the literature, all of which should be classed as carotid body type tumors, from the histological description. He suggests the designation of tympanic body tumors. This develops from the glomus at the jugular bulb and invades the tympanic cavity. It may present in the external auditory meatus and be mistaken for an aural polyp, or grow backward invading the mastoid and destroying the cells. While histologically benign, it has a tendency to recur. Because of its benign character and slow rate of growth, operative treatment may be restricted to radical mastoidectomy with removal of the tumor in the tympanic cavity. Because of the tendency to recurrence patients must be kept under observation and direct attack on the jugular bulb may be required.

HILL.

Plight of the Nerve-Deaf Patient: The Uselessness of all Present Therapy; the Practical Usefulness of Aural Rehabilitation.

Morrissett, L. E.: Archives of Otolaryng. 51:1-25 (Jan.) 1950.

The author concludes that reports of improvement in nerve deafness are unconvincing. He sums up his impressions about as follows: The background of reported successes in the treatment of nerve deafness is of doubtful validity because successes are reported and failures minimized. In many instances, audiometric proof of the supposed improvement does not exist. Even when audiometric details are supplied, criteria of improvement seem inexcusably elastic. Furthermore, posttreatment results are not clearly stated in terms of time. The period of observation is often not long enough to permit reasonable conclusions.

The author discusses several individual studies. The good results reported by Davis and Rommel with neostigmine have not been duplicated by others. Schluederberg reported improvement in 5 patients of 38 treated with neostigmine, but all of these patients also had tinnitus. In another study by Schluederberg, Houser and Campbell, consisting of 56 similarly treated patients, there was no improvement in deafness in a single one, although 7 thought there was some lessening of tinnitus.

There have been many reports on treatment by vitamins, although controlled series of cases are few. The author did not find convincing evidence that vitamin therapy was of any value. Shambaugh and Jennes treated 6 patients with thiamin hydrochloride for 6 weeks and observed no improvement.

Jacobson reported improvement in hearing in 61 per cent of a group treated with benzyl cinnamate. He did not, however, supply any audiometric details.

The author poses the question of the needs of the nervedeaf patient and lists the following as what he must have: (1) complete medical and otologic study, (2) audiometric tests, (3) psychologic studies if indicated, (4) provision of a hearing aid, (5) instruction in lip reading, (6) auditory training, (7) voice training with speech correction if indicated, (8) speech insurance and (9) vocational guidance.

HILDING.

New Neurosurgical Approach for Treatment of Otosclerosis.

Bordes-Valls, Manuel: Archives of Otolaryng. 51:96-102 (Jan.) 1950.

The author describes his technic for a neurosurgical approach to the middle ear for the purpose of fenestrating the labyrinth in otosclerosis. The approach is through the tegmen tympani via the middle cranial fossa. The author removes the tegmen and makes a fenestra in the anterior portion of the lateral semicircular canal. He claims that the operation is much less time consuming and is much easier on the patient. It is a little difficult, however, to see how the purpose of the operation can be accomplished. He does not connect the fenestra in any way with the tensor tympani nor does he cover it by a flap. In the lite of past experience, one would expect this fenestra to close and would not expect any great improvement in hearing.

HILDING.

Interpretation of Hearing Tests.

Guild, Stacy R.: *J. A. M. A.* 142:466 (Feb. 18) 1950.

This paper is primarily concerned with conduction deafness. The principal routes are reviewed along which air-borne sound is transmitted to the inner ear, and the author points out the large number of anatomic structures that participate in its normal transmission across the middle ear. We are reminded that the ratio of air conduction time and bone conduction time is affected by all conductive lesions, but with the less severe lesions, the ratio may be decreased rather than reversed. The possible presence of the conduction lesion is therefore not ruled out in consideration when the answer to the qualitative test is that the fork sounds louder by air conduction than by the bone conduction. The impairment of thresholds by air conduction in each of the conditions that cause conduction deafness may not only differ in amount from slight to marked, but the impairment may be approximately equal for all tones, may be greater for low than high tones, or may be greater for high than low tones. The latter variant, more impairment for high than low tones, can occur with the simplest of all forms of unquestionably pure conduction deafness, that caused by occlusion of the external auditory canal by cerumen or by an obturator, such as an ear plug. This fact alone is sufficient reason for clinical otologists to discard the classic concept, which is still being repeated in textbooks, that a greater loss of hearing for high tones than for low tones always means a nerve deafness. Photomicrographs of the lesions in four cases accompanying the air conduction audiogram in the four ears are used to illustrate the author's point. These are used to emphasize the author's conclusion that otologists in spite of many advances in methods of testing hearing, must continue to rely chiefly on the clinical history and the physical examination in order to determine why a patient is hard of hearing and what can or should be done about it.

BOIES.

Discussion of the Surgical Treatment of Otosclerosis.

Miodonski, J., and Gans, H.: Acta Oto-Laryng. 38:159 (Apr.) 1950.

In reviewing the results of 100 cases of otosclerosis upon whom fenestration had been performed, 76 per cent were reported as gaining improvement in hearing. The addition of 5 cases reoperated upon brings the percentage showing improvement to 81. Improvement was greater in the lower frequencies and varied from 10 to 40 db. Unfortunately the degree of improvement in hearing conversation was not mentioned, the date being compiled from tuning-fork tests and audiograms, as well as the "water curve" test suggested by Miodonski. While persistance of the fistula test generally was found in the improved cases, this was not uniformly so. In a few cases the authors employed "ebesol," a copper perparation, intravenously in the first two post-operative months in an effort to prevent bony closure of the fenestra but have no proof of the effect of this procedure.

HILL.

The Pfeiffer Bacillus in Otitis in Children.

Bjuggren, G., and Tuneval, G.: Acta Oto-Laryng. 38:130 (Apr.) 1950.

In a study of 178 children with suppurative otitis media the Pfeiffer bacillus was found to be the primary agent in 7.3 per cent, as a secondary invader in 3.4 per cent, and either primary or secondary in 1.7 per cent. It was found chiefly in children under four years of age, which group, it is felt have not as yet acquired immunity to this organism. These cases present rather characteristic pictures. The discharge was viscous. While the condition generally responded well to sulfonamide or penicillin therapy there was a marked tendency to relapse and the middle ear was slow to resolve. This might be explained by the tendency of the Pfeiffer bacillus to remain in the nasal cavities after the acute process in the ear seemingly had subsided.

HILL.

Meningocele of the Temporal Bone.

Koch, H.: Acta Oto-Laryng. 38:59 (Feb.) 1950.

The author briefly reviews the literature on meningocele invading the temporal bone and reports a case in which myringotomy was followed by meningitis. There was a discharge of cerebro-spinal fluid from the ear lasting 24 hours. Pneumococci type 11A were found both in the cerebro-spinal fluid and the secretion from the ear. The patient had had symptoms of acute meningitis on several occasions during the previous ten years. It was felt that she had a congenital meningocele in the middle ear which was perforted in performing myringotomy. X-ray examination seemed to confirm this. Recovery was uneventful despite any definitive therapy.

HILL.

NOSE

The Effect of Nasal and Sinus Surgery Upon the Manifestations of Allergy.

Weille, F. L.: New England Journ. Med. 242:43-48 (June 12) 1950.

The author states that nasal and sinus operations usually are satisfactory in the treatment of patients with asthma and vasomotor rhinitis. Asthma of intrinsic, reflex, or extrensic-infection type is reduced in almost half the cases. Slight extrensic-infective asthma in young people often disappears when normal breathing is restored. Obstructive vasomotor rhinitis may be relieved but recurrence of polyposis requires removal.

It is difficult to predict which patients will benefit from surgery and radical measures are not to be undertaken unless there be definite clinical indications. The author feels that when surgical measures are resorted to only in the most severe cases, it is to be expected that the result may be disappointing. He does not consider that asthma is made worse by operation but that it would become worse whether operation was done, or not.

Surgery should be directed toward improved nasal function, anatomical and physiological, and lessening sinus involvement. This may vary from removal of polypi or electrocoagulation of the interior turbinates to submucous resection of the septum, intranasal ethmoidectomy or antrostomy, or the Caldwell-Luc procedure. Rarely is external frontal sinus surgery indicated.

Operations in the sinuses will not relieve nasal obstruction in patients with vasomotor rhinitis. They should not be undertaken for that purpose but to relieve an existing sinusitis.

The author proposes the hypothesis that a specific allergy virus or nasal viral infection can cause nasal and sinus polyposis, vasomotor rhinitis and intrinsic asthma.

This contribution should not be interpreted as advocating surgery while ignoring allergic therapy. It would be unfortunate if such an impression should lead to the formerly too frequent indiscriminate operations upon the nose and sinuses in allergic patients. One might well wish the author had stressed more strongly that these procedures were adjunct to allergic therapy and undertaken only on definite clinical indications.

HILL.

Complications Following Adenoidectomy in Hemophilia.

Walander, A.: *Arch. Oto-laryn.* 36:5-430 (Oct.) 1949.

The author reports a case of unsuspected hemophilia in which adenoidectomy was followed by persistent hemorrhage. Bleeding time was considered to be within normal limits but coagulation time was found to be over 24 hours. Hemorrhage was finally controlled by use of the postnasal pack. Otitis media ensued as a complication despite chemo and antibiotic therapy, requiring three months hospitalization. Subsequent inquiry revealed a previous history of stubborn bleeding following trivial injuries. This case emphasizes the importance of a careful history which so often gives information of more value than routine laboratory procedures.

HILL.

Mucoceles of the Fronto-Ethmoidal Sinuses.

Wigh, Russell: *Radiology* 54:579-589 (Apr.) 1950.

Roentgen findings in eight cases of mucoceles of the frontoethmoid sinuses are tabulated. A short history and roentgenograms of the essential findings are given in each case. Roentgen changes are analyzed and the weight of each in differential diagnosis is considered. The combination of features specific enough to warrant roentgen diagnosis of mucocele is emphasized. Evidence is presented to indicate that some mucoceles, supposedly of frontal sinus origin, begin in anterior ethmoid cells and in fact may be in the frontal bone lateral and separate from the frontal sinus proper.

JORSTAD.

LARYNX

Conservative Surgery in Cancer of the Larynx.

Despons, J.: Revue de Laryngologie, Otologie et Rhinologie 70:418-423 (Sept.-Oct.) 1949.

The author reports five cases of carcinoma of the larynx, from Bordeaux, operated by laryngofissure (thyrotomy). He used the subperichondrial method of St. Clair Thomson. Preliminary tracheotomy was performed in all cases, and the trachea packed off above the cannula. The cannula was removed on the first evening of the following day. Penicillin, 50,000 units, every three hours was given for several days prior to operation, and for six days following the operation. The author stresses the importance of penicillin in preventing secondary infections, promoting early healing, and in preventing pulmonary postoperative infections. The site of the removed block of tissue, within the larynx was packed with iodoform gauze. The gauze was removed with the tracheal cannual or coughed out through the mouth.

The choice of cases, reported here for laryngofissure, does not agree with that generally accepted in the United States. One case was described as presenting a cauliflower-like growth involving almost all of one ventricular band; another case presented a cauliflower-like growth involving the entire left half of the larynx; a third case presented a lesion involving the entire length of the left vocal cord, the anterior commissure, the left ventricular band and left subglottic tissues. In three of the cases, fixation of the vocal cord involved, was noted. The period of follow-up is short: less than a year. All cases survived the operation; in one case a massive recurrence was noted ten months later.

HARKINS.

Radiotherapy of Early Cancer of the Larynx.

Cutler, M.: J. A. M. A. 142:957 (Apr. 1) 1950.

Five year results in 156 cases are reported. These were consecutive cases with the diagnosis proven by biopsy. There were 37 per cent five year cures. More than half of the lesions were considered to be inoperable. In 48 cases in which the lesions were too advanced for laryngofissure and for which the only surgical alternative was total laryngectomy, there were 57 per cent five year cures. The inoperable group yielded 17 per cent cures. Among 58 cases in which the cords were not completely fixed there were 71 per cent cures. The author concludes that radiotherapy is indicted in select cases. When the cords are freely movable or only partly fixed, curability is high and radiotherapy is the method of choice. Total laryngectomy is reserved for advanced lesions with complete fixation of the cords occurring in surgical subjects with a good life expectancy, provided the fixation is caused by the carcinoma. The most important contribution of radiotherapy to laryngeal cancer relates to the group of lesions beyond th escope of laryngofissure, for which total laryngectomy is the only surgical alternative. When such lesions are not yet completely fixed by carcinomatous invasion, they are ideally suited for radiotherapy and the curability is at least equal to that of total laryngectomy.

Boies.

Tracheobronchial Aspiration with a Urethral Catheter—Method of Treatment and Prevention of Asphyxial Hazard in Medical Diseases and Emergencies.

Cardon, L.: *J. A. M. A.* 142:1039 (Apr. 8) 1950.

The author states that a frequent but usually unrecognized complication of many of the conditions that the internist and general practitioner meet in their daily practice consists of obstruction of the airway by accumulating tracheobronchial secretions. Recognition of the presence of these retained tracheobronchial secretions and institution of the appropriate measures to effect their removal without delay should be regarded by the attending physician as his responsibility.

Asphyxia by accumulating tracheobronchial secretions is a frequent complication and may cause certain symptoms and death in many common medical diseases and emergencies, such as pneumonia, myocardial infarction, acute pulmonary edema, acute bronchiolitis superimposed on chronic pulmonary disease, barbiturate and morphine coma, diabetic and uremic coma and cerebrovascular accidents. Aspiration of the tracheobronchial tree is an indispensable procedure for clearing the airway of such secretions.

Endotracheal catheterization and suction is described as a simple method of tracheobronchial aspiration that is immediately available to all internists and general practitioners and can be repeated as often as indicated. It is accomplished by passing a urethral catheter through the nose and into the trachea; in most cases it can be done without direct visualization and without difficulty. No evidence of laryngeal trauma attributable to this procedure has been encountered. The author reports 10 cases illustrating the immediate life saving effect of this procedure.

Boies.

BRONCHI

Bronchoscopy in the Primary and Secondary Period of Tuberculosis.

Fouquet, Jean: Bronchoscopie, Oesophagoscopie et Gastroscopie, pages 7-12, 1949.

The author states that the tracheobroncheal manifestations of the primary and secondary periods of tuberculosis in the infant are only recently recognized. Four types of clinical manifestations are described.

I. The tubercle in the wall of a bronchus: at first this exists as a small tubercle in the bronchial wall, like a chancre. It may soften and become ulcerated, or may become encysted. It may obstruct the bronchus.

II. Manifestations of lymph node infection are mechanical, from pressure on the bronchus, and inflammatory, with congestion of the adjacent bronchial mucosa. Nodes may break down and rupture into an adjacent bronchus, with a persistent draining fistula. Obstruction of the bronchus may occur from the caseous discharge itself, or from the swollen edges of the fistula. This is likely to be a long continued process, leading to changes in the lung, distal to the lesion. A common site of such a fistula is at the carina and on the inner aspects of the main stem bronchi. The course of the fistulae is shortened, by the use of Streptomycin, in contrast with Strepomycin therapy in closed lymph node tuberculosis.

III. Epituberculosis manifests itself by edema of the bronchial mucosa, with production of a quantity of mucous. These act together to partially obstruct the lumen of the bronchus. In general, this process subsides after several months. One rarely, if ever, finds the tubercle bacillus in the bronchial secretions, in these cases.

IV. Other bronchial lesions of primary tuberculosis comprise tuberculoma and polypoid tumors, both causing bronchial obstruction.

HARKINS.

Aerosol and Micronized Ephedrine and Penicillin Therapy of Diseases of the Lower Respiratory Tract. Treatment of Bronchitis, Bronchiectasis and Intrinsic Asthma.

Weinberg, Samuel J., and Packer, George L.: Archives of Internal Medicine 84:395 (Sept.) 1949.

Comparative therapeutic studies on each of two series of 14 patients with chronic bronchial infection, chiefly with bronchiectasis, emphysema and intrinsic asthma, are summarized as follows:

1. The patients in the series treated for three weeks with nebulized (aerosol) ephedrine sulfate U.S.P. followed by aerosol of penicillin calcium gained considerable benefit, as indicated by reducted volume of sputum, increased exercise tolerance, reduced dyspnea, cough and chest discomfort, improved sleep and appetitie and decreased epinephrine requirement.

2. The patients in the series treated for an equal period with micronized (dust) ephedrine sulfate U.S.P. followed by micronized penicillin calcium derived apparently equal benefit in the same details, with an additional increase in vital capacity. Minor reactions, such as oral dryness, saccharin taste and nausea, appeared in this group.

3. Allergic manifestations rarely accompanied the therapy. Inhalation of dust ephedrine will frequently replace the hypodermic administration of epinephrine for asthma.

4. Bacteriologic studies indicated approximately equal effectiveness of moist and dry therapy.

McMahon.

Bronchial Adenoma.

Moersch, N. J., and McDonald, J. R.: J. A. M. A. 142:299 (Feb. 4) 1950.

The observations which form the basis for this paper were made in review of the data on 86 consecutive patients seen at the Mayo Clinic in whom the diagnosis of adenoma of the bronchus was established by microscopic examination of tissue removed from the tumor.

Adenoma of the bronchus should be considered a carcinoma of a low degree of malignancy that possesses the ability to metastasize. It is a disease that occurs with equal frequency in both sexes. The greater majority of adenomas occur in the larger bronchi, although they may occur in any portion of the bronchial tree in which mucous glands are present.

Adenomas may be divided into the carcinoid and cylindroma types. Carcinoid adenomas are the commoner. Cylindromas as a rule have a wider base of attachment and present a more difficult therapeutic problem than do adenomas of the carcinoid type. On the basis of the experience of the cases recorded, it is believed that in suitable selected cases, surgical extirpation of the lesion is followed by the most satisfactory results. The surgical treatment to be employed must necessarily vary, depending on the situation of the lesion, the degree of attachment of the lesion to the bronchial wall, the age of the patient and the degree of secondary suppuration. An adenoma which is pedunculated and situated in a bronchus from which its removal can readily be accomplished is best treated bronchoscopically. Patients of advanced years and those whose lesion is situated close to the carina, so that a pneumonectomy would have to be performed, are best treated by bronchoscopic means. After bronchoscopic treatment has been employed, it is necessary that the patient be subjected to repeated bronchoscopic examination because of the possibility of recurrence.

Surgical treatment is advisable in all other cases and in cases in which the tumor shows evidence of recurrence.

BOIES.

Surgical Treatment of Bronchiectasis in Children.

Buckles, M. G.: J. A. M. A. 143:344 (May 27) 1950.

The diagnosis of bronchiectasis is made only by means of roentgen examination with the injection of a contrast medium, such as iodized oil, into the bronchi. All five lobes should be adequately filled.

The definitive treatment is surgical removal of the suppurative process. The best results are obtained when this removal is carried out early. Twenty-five consecutive cases of bronchiectasis in children treated by pulmonary resection are reviewed. It is a safe procedure as indicated by no mortality in this group.

BOIES.

Tobacco Smoking and Bronchiogenic Carcinoma.

Wynder, E. L., and Graham, E. A.: J. A. M. A. 143:329 (May 27) 1950.

Three independent studies have been made which come to these same conclusions. They indicate that excessive and prolonged use of tobacco, especially cigarettes, seems to be an important factor in the induction of bronchiogenic carcinoma.

The occurrence of carcinoma of the lung in a male nonsmoker or minimal smoker is a rare phenomenon (2.0 per cent).

Ninety-six and one-tenth per cent of patients with cancer of the lungs who had a history of smoking had smoked for over twenty years. Few women have smoked for such a length of time, and this is believed to be one of the reasons for the greater incidence of the diseases among men today.

There may be a lag period of ten years or more between the cessation of smoking tobacco and the occurrence of clinical symptoms of cancer.

BOIES.

ESOPHAGUS

Is the Attempt at Radical Operation for Carcinoma of the Esophagus Successful?

Dick, Walter: Neue Medizinische Welt 1:265-270 (Feb. 25) 1950.

The author, writing for the general practitioner, summarizes the surgical treatment of carcinoma of the esophagus, and emphasizes the fact that there is hope for cure in early cases. He wishes to awaken in his readers an attitude of suspicion for esophageal cancer, in the hope that early lesions will be detected at a stage where cure is possible. Difficulty in swallowing, pain, burning or stinging sensation are the earliest symptoms, suggesting a carcinoma of the esophagus.

The historical development of esophageal resection is traced. The early work of such famous surgeons as Czerny, Billroth, Mikulicz, Sauerbruch, and others, is briefly described. Torek, in 1913, actually performed the first successful esophageal resection, although his patient had to be fed through a gastrostomy opening. Hacker, in 1913 was the first to use successfully the method of anastomosing a segment of large bowel to the upper and lower stumps of the esophagus after resection of a portion. The author examined this same patient 36 years later and found the new food passage to be working well.

Kirschner, in 1920, anastomosed the stomach to the upper esophageal stump, after resection of the esophagus, and actually perfected the technical problem of resecting the thoracic portion of the esophagus for cancer; he constructed a tube of skin to serve as a substitute for the removed portion of the esophagus.

The author states that in recent years, successful resections of the esophagus are being done in what he calls the Anglo-American countries. He cites the work of Garlock, who in 1946 reported over 250 such operations with surprisingly good results. The author himself has performed six resections in the last few years; two patients died: one, several weeks postoperative of coronary thrombosis, the other of cerebral lues. The other four survived and are able to swallow normally.

HARKINS.

MISCELLANEOUS

The Question of Occult Mastoiditis in Nursing Infants.

Sauerbrei, Hans Ulrich: *Neue medizinische Welt* 1:206-209 (Feb. 11) 1950.

Occult mastoiditis is a concealed type of infection—a retrotympanic, destructive osteitis. It can not be detected from external examination of the ear. It is characterized by a severe, acute toxic disturbance of nutrition of the nursing infant, with marked loss of weight, refusal to eat, and vomiting. It does not present the usual otorrhea. This entity was first described in the German pediatric literature in 1928 by Finkelstein, but had been previously described in the United States by Marriott, as "masked otitis." The infection spreads out from the antrum into the not yet pneumatized mastoid process; the infection is a destructive process, yet remains without local manifestations.

As diagnostic signs, the author lists: sudden cessation of a previously existing otorrhea, sagging of the upper posterior wall of the external auditory canal, tenderness to pressure over the mastoid process, edema over the mastoid, slight redness of the ear drum. There may be no local signs, whatsover. There is a sudden exacerbation of general symptoms, such as refusal to eat, vomiting, diarrhea, loss of weight. Toxic symptoms occur, such as stupor, meningismus, alternating periods of restlessness and apathy. There is no characteristic fever curve; fever occurs in only half of the cases. Usually, there is leukocytosis with a "shift to the left" in the differential count. In arriving at a diagnosis appropriate steps are taken to rule out other acute infections, such as pneumonia, meningitis, pyelitis.

The author strongly recommends bilateral mastoid antrotomy, as early as possible after the diagnosis is made. Supportive measures, such as parenteral fluids, blood transfusions, sulfonamides or penicillin, are advised, but can not take the place of surgical eradication of the infected focus in the mastoid process.

HARKINS.

Chipped Glass as a Probably Cause of Retropharyngeal Abscess in an Infant.

Gardner, L. I., and Heinicke, H. I.: *New England Journ. Med.* 242:25-975 (June 22) 1950.

The authors report a case of retropharyngeal abscess in an infant, apparently due to ingestion of a piece of glass from a jar. Two incisions evacuated pus and gas, culture giving a growth of Escherichia coli. Streptomycin was added to the initial therapy of penicillin and sulfadiazine. The patient was discharged on the 20th hospital day.

HILL.

The Effect of Streptomycin Medication on the Galvanic Reaction.

Ingelstedt, S., and Walander, A.: Arch. Oto-laryn. 37:6-523 (Dec.) 1949.

The toxic effect of streptomycin upon the vestibular reaction generally has been determined by rotation and caloric tests. The authors consider that the galvanic test might be of prognostic value; that if this is normal the prognosis is good, but recovery is doubtful if response to the test is lost. They feel that the lesion is in the vestibular ganglion and in Deiter's nucleus, and that these nerve cells are quite incapable of regeneration.

HILL.

Frontal Osteomyelitis. Its Treatment and Some Experimental Observations on the Effect of Heparin in Combination with Sulphonamide and Penicillin Therapy.

Herberts, G.: Arch. Oto-laryn. 37:4-321 (Aug.) 1949.

From a study of six cases of frontal osteomyelitis and one of osteitic involvement of the maxilla complicated by meningitis and cavernous sinus thrombosis, the author concludes that early surgical drainage combined with penicillin therapy may make unnecessary more radical surgical measures. Penicillin has the capacity of penetrating fibrin, and also blood coagulum, which the sulphonamides are unable to do. Because of the importance of infected thrombophlebitis, in the pathogenesis of osteomyelitis anticoagulants should be combined with sulphatherapy in cases where the organism is resistant to penicillin. This also increases the bacteriostatic effect of sulpha. The author feels that this combined therapy is more effective in severe infections with impaired blood supply due to thrombosis.

HILL.

Dacryocystorhinostomy: Report of One Hundred and Fifty Cases of Dacryocystitis Occurring from 1938 to 1947.

Welt, Bernard: Archives of Otolaryng. 51:83-95 (Jan.) 1950.

The author gives an historical review of the attempts to cure obstruction of the lacrimal sac by surgical means. He then gives the results in 150 cases treated by the operation of dacryocystorhinostomy. His results are good and seem to parallel the experience of ophthalmologists who use this operation. He describes his technic in some detail, adding several new refinements of his own. He makes the bony window large and removes completely any ethmoid cells encountered. He states that failures are due to osteogenesis and to infection and that osteogenesis can be prevented by making the window large enough.

(Reviewer's comment: If the tear sac can be anastamosed to the lining of an ethmoid cell, it would presumably be just as satisfactory as anastamosing it to the lining of the nasal chamber. Osteogenesis does not depend upon the size of the window made in a bone but rather upon the accuracy and completeness with which the new tract is lined by epithelium.)

HILDING.

Peripheral Facial Paralysis in Fractures of the Temporal Bone: Indications for Surgical Repair of the Nerve; Report of Cases in which the Balance and Duel Operation was Used.

Karsten, Kettel: Archives of Otolaryng. 51:25-42 (Jan.) 1950.

The author divides fractures of the temporal bone into two main types, longitudinal and transverse, of which the longitudinal is by far the more common and has the better prognosis both as to life and cochlear and vestibular function.

In the cases of longitudinal fractures which the author studied, facial paralysis was present in from 10 to 18 per cent and in the transverse fractures in about 50 per cent. He divides the paralysis into two groups, those occurring immediately and those occurring after some time has passed. The prognosis is favorable with conservative treatment in those having delayed paralysis, whereas the prognosis of those showing paralysis immediately is not so good. Twenty-five per cent of the latter studied by Turner did not clear up spontaneously.

The author admits that there are difficulties in determining which cases should be selected for operative repair of the facial nerve. He concluded that all severe injuries of the facial nerve should be operated as soon as the patient's general state of health permits. Sometimes it is not possible to determine how severely the facial nerve has been injured. In this case, he waits two months and, if there has been no spontaneous return of function in that time, he assumes that the lesion is severe and decompression is indicated. The majority of cases of delayed paralysis show signs of returning mobility within two months. Should this not be the case, treatment is similar to that for immediate paralysis and it is assumed that a severe injury of the nerve has occurred and that decompression is indicated.

In cases of long-standing paralysis, with very little or no facial function, an exploration should be done provided the muscles have not undergone atrophy and provided the site of the damage can be located. If, however, the patient already has partial recovery and the function is fairly good, decompression should not be done because further improvement is problematic. If the place of injury is surgically inaccessible, then an anastamosis between the facial and another cranial nerve should be carried out. The author stresses that these operations should be undertaken only by otologists specially trained for the work. In all cases of facial paralysis, treated either conservatively or surgically, the muscles should be kept alive by means of massage and galvanic stimulation.

HILDING.

Human Tolerance for Large Amounts of Radiation.

Newell, R. R.: Radiology 54:598-601 (Apr.) 1950.

Results are reported of a questionnaire answered by thirty-three presumed experts in regard to tolerance of man for large amounts of radiation (x-rays or gamma rays) over the whole body. Use of these data in times of atomic disasters is discussed. Author concludes that the opinions in regard to human tolerance for large x-ray doses vary so greatly that it appears highly desirable to obtain some dependable data.

JORSTAD.

Trigeminal Injection.

Sweet, W. H.: J. A. M. A. 142:392 (Feb. 11) 1950.

The author recommends the use of radiographs to determine the position of the needle for injection of alcohol into the second and third divisions of the trigeminal nerve. He claims certain advantages for this method as follows: the pain of the procedure is decreased, the number of complete injections into the nerve is increased to over 95 per cent of those attempted, and the average duration of relief from the pain of trigeminal neuralgia following injections in the third division is increased to at least thirty months. This is accomplished by pointing the bevel of the needle upward which he believes allows the alcohol to penetrate up the nerve trunk to the cells of the gasserian ganglion.

BOIES.

Evaluation of Dizziness.

De Weese, D. D.: *J. A. M. A.* 142:542 (Feb. 25) 1950.

A carefully taken history is important in evaluation of dizziness. It is also important that whirling vertigo be distinguished from other sensations described as dizziness by the patient.

Systematized vertigo usually indicates disease of the statokinetic system. Nonsystematized vertigo may arise from disease in any part of the body.

Disease of the proprioceptive, cardiovascular and central nervous system commonly gives rise to dizziness outside the statokinetic system. The author emphasizes a most important observation to the effect that the otolaryngologist should become increasingly more alert to general systemic disease in which dizziness is one symptom.

BOIES.

Anaphylactoid Shock Due to Penicillin.

Burleson, R. J.: *J. A. M. A.* 142:562 (Feb. 25) 1950.

A patient with typical anaphylactoid shock after the administration of penicillin was treated intravenously with diphenhydramine hydrochloride and recovered. Secondary symptoms developed which were probably due to insufficient intravenous injection of diphenhydramine hydrochloride or to the lack of a subsequent injection of the same drug. The author observes that the initial dose should probably have been repeated for a maximum benefit.

This case illustrates the necessity for adequate investigation regarding previous penicillin therapy and atopy from any acuse. Although the incidence of penicillin as an allergic offender has been negligible to the present time, this valuable drug should not be used indiscriminately.

BOIES.

Irradiation as the Preferred Treatment of Cancer of the Lip.

Sharp, G. S., Williams, H. F., and Pugh Jr., R. E.: *J. A. M. A.* 142:698 (Mar. 11) 1950.

The authors contend that complete eradication of cancer of the lip may be accomplished, with minimum resulting deformity, in a high percentage of cases by either irradiation or surgery. The best possible esthetic result is obtained by irradiation.

Judgement for the type of treatment is based on the depth of invasion, the curability of the primary lesion by the simplest method and the best cosmetic result. Radium by the surface plaque or with interstitial needles fulfills these requirements and is most satisfactory in a majority of cases. The cure rate in cancer of the lip is higher than for any other form of oral cancer. The absolute percentage of cures was 84.2 which included all cases lost for follow-up and all deaths from inter-current disease. If these lost cases are excluded as well as those of patients known to be free of cancer of lip when they died, the percentage of patients free of disease five years was 95.7, a closer approximation of the actual prognosis of the disease.

BOIES.

Antihistaminic Drugs.

Sternberg, T. H., Perry, D. J., and Le Van, P.: J. A. M. A. 142:969 (Apr. 1) 1950.

The authors have studied the comparative activity in man of the antihistaminic drugs. The activity was measured by histamine iontophoresis.

The method used consists of iontophoreses of serial dilutions of histamine base. The initial threshold is determined by the highest dilution of histamine base producing diffuse punctate whealing at the site of the positive electrode. The drug to be tested is then administered orally and the threshold again determined one and two hours later. The difference between the initial and subsequent readings is the measure of the antihistaminic activity of the test drug.

The most active antihistaminic drugs as determined by this method were pyribenzamine, benadryl, and neoantergen. The data also demonstrate a variation in activity in different persons receiving the same antihistaminic drug. These data correlate closely with clinical experience.

BOIES.

Aureomycin in Infectious Mononucleosis.

Seifert, M. H., Chandler, V. L., and Van Winkle, Jr., W.: J. A. M. A. 142:1133 (Apr. 15) 1950.

Infectious mononucleosis is a disease of unknown causation. It is suspected of being of virus origin. No satisfactory treatment exists for it at present. (The otolaryngologist often sees these cases because of the complaint of sore throat.) The course of the disease is usually protracted.

Contrary to general impression, infections mononucleosis may occur at any age, and 19 of the 47 patients were 35 years of age or older. The oldest patient was 59, and 8 were under 12 years of age.

The duration and severity of infectious mononucleosis are extremely variable, and in order to evaluate the effectiveness of a proposed therapeutic agent in this disease it is imperative that an adequate control series be studied simultaneously.

The authors noted an epidemic of this disease in the north suburbs of Chicago during the winter of 1948 and 1949. This epidemic offered an opportunity for a controlled study.

Forty-seven cases of infectious mononucleosis comprised the group. Of this number 26 patients had the clinical picture, blood smears and results of heterophil agglutination tests considered diagnostic. Aureomycin was administered and the frequency of favorable response to this drug and to a placebo was that which could be expected by chance. In a second group of 21 patients in whom the clinical picture and blood smear were considered diagnostic for infectious mononucleosis but in whom the heterophil agglutination test was equivocal or negative, similar results were obtained. In the first group, 17 to 26 patients had cold agglutinin titers of 1:32 or higher without physical observations or symptoms suggesting the presence of a typical primary pneumonia, thus casting some doubt on the specificity of the test.

Toxic effects, principally nausea, vomiting, diarrhea and burning of the rectum were observed in 69 per cent of the patients receiving aureomycin. It should be mentioned, however, that an extensive clinical experience with aureomycin from the same source in recent months demonstrates much less toxic effect from the more highly purified product. No patients receiving the placebo had side effects.

It is obvious from the data presented that aureomycin is of no value in the treatment of infectious mononucleosis. This is contrary to reports of uncontrolled studies which have appeared.

BOIES.

Fatalities Following Curare.

Foregger, R.: *J. A. M. A.* 142:1344 (Apr. 29) 1950.

The use of this drug has become rather widespread in clinical medicine. It contacts the field of otolaryngology chiefly in anesthesia for augmenting relaxation during surgical operations. Three fatalities considered to be due to a dangerous side action of curare are reported. The literature was partially reviewed and found to contain references to 19 fatalities following the use of curare. Several near fatalities are reported. These are likewise considered to be due to the side action of the drug. The incidence of fatal reaction with curare is low. At the present time available knowledge suggests that the antihistamine drugs and procaine may be helpful in treating respiratory complications of curare.

BOIES.

Antihistaminic Drugs for Colds.

Hoagland, R. J., Lt. Col., Deitz, E. N., Capt., Myers, E. W., Lt., and Cosand, H. C., Lt., of the Medical Corps of the United States Army: *J. A. M. A.* 143:157 (May 13) 1950.

This is an evaluation of antihistaminic drugs in a controlled study. The necessity for such a carefully controlled clinical investigation of the treatment of the common cold is again demonstrated; in this investigation 27 per cent of the patients receiving inert materials report a cure within 24 hours.

There was no significant difference in the proportion of cures reported by patients receiving oral antihistaminic drugs and those receiving oral placebos. Essentially, the same proportion of patients received no benefit from either type of treatment.

Patients receiving antihistaminic drugs within 24 hours after symptoms began were as likely to fall into the "no effect" group as into the cure group. The effect of pyribenzamine administered by nebulizer was essentially the same as the effect of inert materials given orally and intranasally.

Although the use of antihistaminic drugs orally and intranasally may decrease the irritating nasal discharge, characteristic in the early stages of the common cold, the brief and variable duration of this phase makes a positive conclusion regarding this point difficult.

BOIES.

Stuttering—The Problem Today.

Karlin, I. W.: *J. A. M. A.* 143:732 (June 24) 1950.

Stuttering is defined as a disturbance in the rhythm of speech. The symptoms unfold gradually. The principal present day theories are that stuttering is: (1) a psychoneurosis or personality disorder; (2) a habit or behavior that is learned; and (3) an organic disorder of the language function. The theory that it is due primarily to a slower process of myelinization of the cortical speech areas offers a satisfactory explanation of the basic facts. Emotional and environmental factors play an important role in unfolding and perpetuating the disorder. Emphasis in the treatment should be on prevention. Every pre-school child who shows early signs of stuttering should receive immediate treatment.

Boies.

Anemia Following Use of Antihistaminic Drugs.

Crumbley, J. J.: *J. A. M. A.* 143:726 (June 24) 1950.

Three cases of hemolytic anemia associated with the administration of antihistaminic drugs are reported. One is a case of bronchial asthma treated with benadryl. The others are cases of hay fever. The two cases of hay fever were treated with pyribenzamine. The drugs were taken over several months of time. In all three cases response to withdrawal of the drug was return to normal of the blood picture. The logical conclusion was that these drugs should not be administered without medical supervision of the case.

Boies.

Antihistamic Agents and Ascorbic Acid in the Early Treatment of the Common Cold.

Cowan, D. W., and Diehl, H. S.: *J. A. M. A.* 143:421 (June 3) 1950.

The effects of antihistaminic drugs and ascorbic acid on the early symptoms of the common cold is reported from a controlled study. Nine hundred eighty colds were treated in 367 students who were all supposedly nonallergic. From this study it is concluded that there is no indication that ascorbic acid alone, phenindamine tartrate (thephorin) alone, ascorbic acid plus phenindamine, or tripelennamine (pyribenzamine) hydrochloride have any important effect on the duration or severity of these infections of the upper respiratory tract.

Boies.

Behcet's Syndrome. Report of a Successfully Treated Case.

Koch, H.: Acta Laryng. 38:56 (Feb.) 1950.

Behcet, in 1937, reported two cases of recurrent aphthous stomatitis of long duration, asociated with lesions on the eyes and genitalia. He considered it due to a virus but this has now been proven. Various therapeutic procedures have been futile. The author presents a case which had, in addition, symptoms of chronic colitis and in which the gastric symptoms proceded the oral manifestation. For 20 years the patient had had recurring painful ulcerations in the mouth, soft palate, tongue, and fauces, together with a keratoconjunctivitis and vesicles on his scrotum. Treatment with "antastin," considered to be specific for allergic conditions, resulted in rapid improvement. From this the author feels that possibly Behcet's syndrome is an allergic disease and suggests the desirability of thorough allergic studies in such cases.

HILL.

Books Received

Some Relations Between Vision and Audition.

By J. Donald Harris, Ph.D., Head, Sound Section U. S. Naval Medical Research Laboratory, New London, Connecticut. Pp. v +56. Springfield, Illinois, Charles C. Thomas, 1950. (Price $1.50)

Office Treatment of the Nose, Throat and Ear.

By Abraham R. Hollender, M.Sc., M.D., F.A.C.S., Professor of Otolaryngology, Emeritus, University of Illinois College of Medicine; Attending Otolaryngologist, St. Francis Hospital and Mt. Sinai Hospital, Miami Beach; Consulting Otolaryngologist, Variety Children's Hospital, Miami, Florida. Pp. 620, with 146 illustrations. Chicago, Illinois, The Year Book Publishers, Inc., 1950. (Price $7.50)

Oral and Facial Cancer.

By Bernard G. Sarnat, M.D., F.A.C.S., Professor and Head of the Department of Oral and Maxillofacial Surgery, College of Dentistry, and Clinical Assistant Professor of Surgery, College of Medicine and Research and Educational Hospital, University of Illinois, Chicago; Diplomate of the American Board of Plastic Surgery, and Isaac Schour, D.D.S., Ph.D., Sc.D., Coordinator of Cancer Instruction, Professor and Head of the Department of Histology and Associate Dean in Charge of Postgraduate Studies, University of Illinois College of Dentistry, Chicago. Pp. 300, with 118 illustrations, and 2 tables. Chicago, Illinois, The Year Book Publishers, 1950. (Price $6.00)

Carcinoma of the Larynx.

By F. Baclesse, M.D., "Chef de Service" of the X-ray Department Foundation Curie, Paris. With A Statistical Report on Cases Treated with X-rays Only, By R. W. Gunderson, B.Sc., D.M.R.T., F.R.C.S., (Eng.) from The Meyerstein Institute of Radiotherapy, and The Middlesex Hospital, London, W. I. Pp. 68, with 97 illustrations, and 20 tables. London, W. I., The British Institute of Radiology, 1949. (Price 25s)

Cytologic Diagnosis of Lung Cancer.

By Seymour M. Farber, M.D., Milton Rosenthal, M.D., Edwin F. Alston, M.D., Mortimer A. Benioff, M.D., and Allen K. McGrath, M.D. From the University of California Medical Service and Department of Pathology, San Francisco Hospital and the San Francisco Department of Public Health. Pp. xii+79, with 60 illustrations on 10 plates in color. Springfield, Ill., Charles C. Thomas, 1950. (Price $6.00)

Supervoltage Roentgentherapy.

By Franz Buschke, M.D., Simeon T. Cantril, M.D., Herbert M. Parker, M.Sc. From the Tumor Institute of the Swedish Hospital, Seattle, Washington. Pp. xiii+297, with 150 illustrations, 20 in color, and 19 tables. Springfield, Ill., Charles C. Thomas, 1950. (Price $10.50)

Chemotherapie der Tuberkulose mit den Thiosemikarbazonen.

By Prof. Dr. G. Domagk, Wuppertal-Elberfeld. Unter Mitarbeit von C. Arold, K. Boshamer, H. Delfs, L. Heilmeyer, K. W. Kalkoff, Ph. Klee, R. Knorr, F. Kuhlmann, A. Lemberger, H. Malluche, K. Ullmann. Pp. viii+405, with 206 illustrations. Stuttgart, Georg Thieme Verlag, 1950. (Price $14.30)

The Esophagus and Pharynx in Action.

By William Lerche, M.D., Fellow, American College of Surgeons, Founder Member and Honorary Member of the American Association for Thoracic Surgery, Formerly Associate Professor of Surgery University of Minnesota, Minneapolis, Minnesota. Pp. xii+223, with 93 illustrations, and 5 tables. Springfield, Illinois, Charles C. Thomas, 1950. (Price $5.50)

Notices

A. A. A. S.

The 117th Meeting of the American Association for the Advancement of Science, the annual meeting for the year 1950, will be a full-scale meeting with programs in every principal field of science from astronomy and botany to, and including, zoology. All 17 of the Association's sections and subsections, and more than 40 participating societies and organizations, are completing plans for an aggregate of more than 200 sessions.

Announcements and coupons for reservations will appear in Science and The Scientific Monthly, beginning the last of August. The hotels include the Statler (headquarters of the AAAS) the Hollenden, the Carter, the Allerton, the Olmsted, and the Auditorium Hotel—the last particularly convenient for exhibitors.

Plans are already made to ensure the mailing of the General Program to advance registrants not later than December 1 (as compared with December 6-9 last year) *by first class mail.*

Raymond L. Taylor,

Assistant Administrative Secretary

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Washington, D. C.

HARVARD MEDICAL SCHOOL

The Harvard Medical School offers a series of four consecutive graduate courses in otology and laryngology from November 6, 1950, to March 31, 1951.

Courses will be given in Histopathology, Audiology, Anatomy of the Head and Neck and Anatomy of the Temporal Bone.

For further information, apply to Assistant Dean, Courses for Graduates, Harvard Medical School, Boston 15, Massachusetts.

AMERICAN BOARD OF OTOLARYNGOLOGY

The American Board of Otolaryngology will conduct the following examinations:

October 3-6 in Chicago, Illinois, at the Palmer House.

January 8-11 in New York City, at the Hotel Biltmore.

DEAN M. LIERLE, M.D., *Secretary.*